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STUDIES ON THE ASCARIS LUMBRICOIDES

BY

HARRY MATHIAS MARTIN

A THESIS

PRESENTED TO THE FACULTY OF THE
GRADUATE COLLEGE OF THE UNIVERSITY OF NEBRASKA
IN PARTIAL FULFILLMENT OF THE REQUIREMENTS
FOR THE DEGREE OF DOCTOR OF PHILOSOPHY
DEPARTMENT OF ZOOLOGY

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Studies on the Ascaris Lumbricoides

H. M. MARTIN
Department of Animal Pathology and Hygiene

LINCOLN, NEBRASKA
Accepted for publication June, 1926
December, 1926

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Studies on the Ascaris Lumbricoides

H. M. MARTIN

INTRODUCTION

The *Ascaris lumbricoides,* in one phase or another, has attracted the attention of parasitologists for many years, and during the last decade this parasite has been studied by a large number of investigators. The recent observations have been, to a great extent, directed along one line, the study of its life history. These investigators have established two important points: (a) That the life history of *Ascaris* is more complex than was supposed, as it has been found that a vasculo-pulmonary circuit of the larvae is necessary before they settle down in the intestine, where they develop to the adult stage; (b) that this parasite may not produce its greatest damage while in the intestine of swine or man. It is capable of exerting a more harmful effect during the interval between hatching in the intestine and its return to the intestine to develop to the adult stage. During this period all organs of the body may be involved, especially the lungs and liver.

Other phases of the *Ascaris* problem, such as the probable identity of the human and pig *Ascaris,* the question of intrauterine infection, parasitic allergy, immunity, etc., have been investigated and discussed by a number of workers.

The great interest of farmers and swine raisers in this parasite induced the writer to undertake certain investigations along the line mentioned above, the results of which are here reported.

HISTORICAL REVIEW

It appears from the evidence presented in literature that Küchenmeister (48), in 1855, was the first to undertake feeding experiments with embryonated eggs. He experimented with a young dog, and, from his discussion, apparently obtained negative results. Mosler's (63) experiments, in which he fed *Ascaris* eggs to human beings, failed to produce *Ascaris* in the intestinal tract. These failures, therefore, led him to suspect that temporary hosts or carriers transmitted this parasite to man. Leuckart (51), after having obtained negative results in all of his experiments on various animals, therefore conjectured that the embryos must get out of their shells and live freely for a time or that the life cycle must be completed by a change of host, and regarded the latter

hypothesis the more probable. He suspected lower animals to be the intermediary hosts and conducted a large number of experiments on invertebrates searching among them for the temporary host. Von Linstow (52), in 1886, thought he had found this intermediate host, and described it as being *Julus guttulatus,* a small myriapod in the intestine of which *Ascaris lumbricoides* eggs were found.

The hypothesis of an intermediate host being necessary to complete the life cycle of *Ascaris lumbricoides* was disproved by Grassi (36-37), Lutz (57), and Epstein (23), who, without doubt, produced direct infection in man by administering eggs in which the embryos had reached the vermiform stage.

Grassi (36) administered a pill containing embryonated eggs to a small boy seven years old, and after about two months *Ascaris* eggs were found in the feces. The child was given an anthelmintic treatment which resulted in the elimination of 143 *Ascaris.* In 1879 he (37) also swallowed 100 eggs with living embryos and later observed eggs in his feces. This experiment apparently was the first in which a direct infection was assumed to have been produced.

Lutz (57) in 1888 reported a successful experiment on the transmission of *Ascaris* to man. He administered embryonated eggs to a 32-year-old patient that was supposed to have been absolutely free of *Ascaris* infection for the last 20 years. The patient was later given anthelmintic treatment, after which 35 immature *Ascaris* were recovered in the feces.

Early in the year of 1891, Epstein (23) administered ripe eggs to three children. The feces of two of these children were examined 74 days after administration of the eggs, at which time the feces were noted to be free of eggs, but 12 days later the feces contained many eggs. The feces of the one six-year-old child were not examined until about six months later, at which time large numbers of eggs were found. Two of the children were treated with an anthelmintic which caused the four-and-one-half-year-old child to eliminate 22 and the second child to eliminate 79 *Ascaris.* With this evidence before us, there is no question but that these experimental children were successfully infested with *Ascaris* by the feeding of *Ascaris* eggs.

In 1916 Stewart (87) made a preliminary report of his work on the life history of *Ascaris* in which he points out that the life history is not so simple as was generally supposed. He made the highly interesting and important discovery that when ripe eggs reach the alimentary canal of the rat and mouse they hatch. The larvae, when liberated, enter

the bodies of their hosts and reach the liver and lungs. During their migration they undergo considerable development.

Davaine (19) in 1863 observed that living larvae can be found in the small intestine of a rat 12 hours after feeding *Ascaris* eggs, but Stewart (87) was the first to observe that in these animals not all the hatched larvae were promptly eliminated in the feces, but that some penetrated the wall of the alimentary canal, and wandered to other parts of the body, during which time they increased in size and underwent other morphologic changes. He (88) observed further that the larvae do not remain in the lungs, but migrate into the bronchi and up the trachea into the mouth of the rat or mouse. These findings led him to suspect that the saliva of rodents containing larvae might possibly contaminate food and in this way transfer the young parasites to pig and man. Stewart (89) in later experiments noted that after the larvae had passed into the bronchi and up the trachea they then were conveyed down the oesophagus and into the intestine, and accumulated in the caecum and large intestine. After arrival in the large intestine, a large number of the larvae are voided in the feces without undergoing any marked change in size or structure from the stage attained in the lungs.

Similar experiments conducted by Stewart (87-90-91-94) on pigs failed to reveal definite evidence that these animals became infected by swallowing *Ascaris* eggs. He therefore offered the hypothesis that rats and mice serve as intermediate hosts in the life cycle of *Ascaris;* that is, they became infected by swallowing eggs passed in the feces of man and pig. In turn they contaminate the food and drinking water consumed by the definite hosts with feces containing larvae that have passed thru the lungs and back into the intestine by way of the trachea and oesophagus.

The hypothesis presented by Stewart on the life history of *Ascaris* caused Lane (50) and Low (53) to present a number of reasons for doubting the possibility of rats and mice acting as intermediate hosts. Ransom and Foster (76-78) have also indicated that the rat and mouse theory is not tenable. The investigations of Ransom and Foster have shown that the larvae will migrate from the intestine to the lungs in the cavia, rabbit, sheep, pig, and goat, the same as in the rat and mouse. They also indicated that, in the definite host, some of the larvae after having returned to the intestine from the lungs may establish themselves and develop to the adult stage. Stewart (95-96-98-99) in later papers reported additional

experiments on young pigs which apparently resulted in the *Ascaris* establishing themselves in the small intestine where they developed from 3.8 to 7 mm. in length in from 14 to 19 days.

Koino (46) in an experiment upon himself was the first to recognize that the *Ascaris* larvae in man will migrate from the intestine to the lungs and up the trachea. He recovered large numbers of larvae in his sputum. He also established the fact that the larvae, after having completed their migration thru the lungs, will settle down and develop to the adult stage.

Yoshido (109) reported results of experiments from which he concludes that the larvae migrate actively by boring thru the tissues to the lungs, after hatching in the intestine, and that the migration of the larvae in the blood stream, if it occurs at all, is of secondary importance. Asada (3) also reports that *Ascaris* larvae bore thru the intestinal wall and gain the abdominal cavity, but instead of boring thru the diaphragm they wander toward the surface of the liver, and from here penetrate into the parenchyma of the liver. He states, however, that he was also able to determine that the larvae gain the liver direct from the intestine by means of the blood stream.

The experiments and observations made by Ransom and Cram (15) are not in harmony with the conclusions reached by Yoshido (109), as their results clearly demonstrated that the larvae are carried from the intestine to the lungs by the blood stream. The researches of Fülleborn (28-30-33), likewise, do not confirm Yoshido's (109) findings. He (30) states that the vast majority of the *Ascaris* larvae which escape from the eggs in the gastro-intestinal tract reach the lungs by way of the blood vascular system and not thru the peritoneal cavity and diaphragm.

It has recently been shown by Danheim (16) that the hatching of *Ascaris* eggs and migration of the larvae not only occur in the body of mammals, but may also occur in birds. She found that, if infectious *Ascaris lumbricoides* eggs are administered to young fowls, they will hatch in the alimentary canal, and the larvae will migrate from the intestine to the liver and lungs the same as in mammals.

Stewart (87) further observed that the larvae not only migrate thru the lungs, but in so doing may produce an intense pneumonia which may prove to be fatal in experimentally infected rats and mice. He (91-94-95-96-98-99) later observed that the migrating larvae will also produce

pneumonia in pigs. Ransom and Foster (76-78) also noted the occurrence of pneumonia as a result of migrating *Ascaris* larvae in rats, mice, and pigs. They (77-78) observed further that a fatal pneumonia may be produced in cavias, rabbits, and young goats as a result of larvae migrating thru the lungs. These findings led to the belief that migrating *Ascaris* larvae may also produce *Ascaris* pneumonia in man. In fact, Mosler (in Leuckart, 51) reported the occurrence of lung symptoms in an experimental child. Lutz (57) also reported symptoms of a severe bronchitis in an adult to whom he had fed the eggs.

The lung symptoms observed by these investigators were undoubtedly associated with the migration of *Ascaris* larvae, altho these workers did not appreciate the significance of the symptoms at the time, as they regarded them as probably resulting from some external cause. In a recent publication Steiner (86) reported a case of pneumonia in a child that he regarded as being due to pulmonary ascariasis.

Koino (46) in conducting experiments on his brother and himself demonstrated conclusively that *Ascaris* larvae will produce *Ascaris* pneumonia in man. In these experiments Koino's brother ingested 500 pig *Ascaris* eggs and Koino himself took 2,000 human *Ascaris* eggs, which resulted in an *Ascaris* pneumonia on the sixth to ninth day after the ingestion of the eggs in both individuals.

THE DESCRIPTION OF THE ADULT ASCARIS LUMBRICOIDES

The coloring of the *Ascaris* in the fresh condition is a reddish-yellow or a grayish-yellow; the body is smooth, and of an elongated, spindle shape. It is marked by numerous fine transverse rings and becomes gradually attenuated toward either extremity, the anterior terminating in a tri-papillated mouth, the posterior in a bluntly pointed tail.

The oral papillae are finely denticulated. The dorsal papilla carries two sensory papillae and the two ventral papillae each one sensory papilla. The oral aperture, without any other special appendages, leads directly into a strong, muscular oesophagus which varies in length in different specimens. A slight constriction separates this organ from the intestine.

The intestine is a yellowish-brown color, which is mainly due to the intestinal contents, altho the epithelium and general cell wall are more or less supplied with dark pigment granules. The intestinal canal in both sexes terminates in a

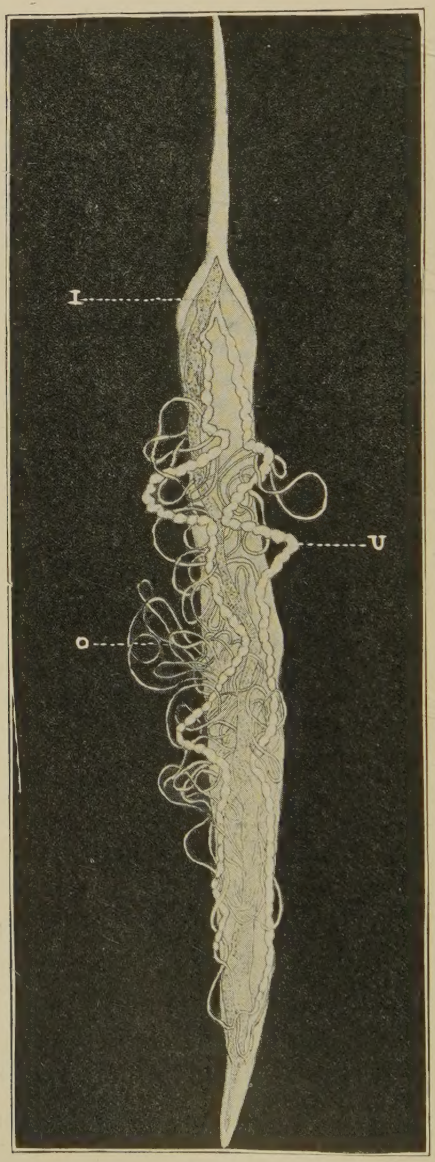


FIG. 1.— Female Ascaris dissected. Half natural size. I, intestines; U, uterus; O, ovary.

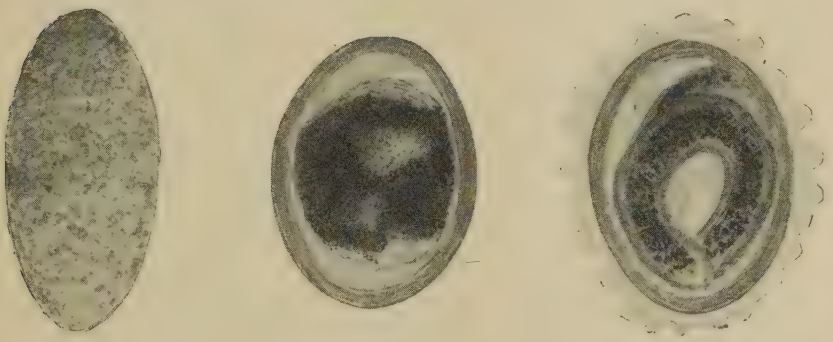
cloacal cavity which opens externally by a transverse slit a short distance from the extremity of the tail. In this situation there are 70 to 75 papillae, of which seven pairs are postanal. The male measures 150 to 250 mm. in length, according to most authors, but the writer has found males measuring 310 mm. in length and from 2 to 4 mm. in diameter; the posterior extremity is conical and bent hook-like ventrally. At the cloaca two small spiculae are found; these spiculae measure about 2 mm. in length, are curved, and are somewhat broadened at their free ends. The testicular tube is about eight times the length of the body, is very tortuous, shows thru the integument, and terminates into a cone-shaped organ, the vesiculus seminalis. In the female, the writer has noted a much greater variation in regard to size than is usually described by other authors. He observed that uteri from *Ascaris* as small as 125 mm. in length contained eggs which were fully developed and fertilized, thus establishing the fact that females of this size may be considered as adults. The writer has also found females which were 490 mm. in length, this being 90 mm. longer than the

maximum length usually given by other authors. The female varies in diameter from 3 to 6 mm. The posterior extremity is conical and straight. The vulva is situated at the junction of the anterior and middle third of the body, which, at this point, has a slight ring-like construction. The convoluted filiform ovaries measure from five to ten times the length of the body and merge into their respective oviducts, beyond which is the receptaculum seminis, which terminates in the bifurcated uterus.

THE EGG STAGE OF ASCARIS

DESCRIPTION OF THE ASCARIS EGG

The ova are elliptical, with a thick, transparent shell and an external albuminous covering which is coarsely mammillated. The ova measure from 39 to 75 microns in length and from 36.4 to 58 microns in breadth. The albuminous covering is sometimes absent from ova which are deposited by the



A B C

FIG. 2.—Ascaris eggs. Enlarged 500 times. a. dead or sterile egg; b. egg taken freshly from the uterus; c. ripe, infective egg with larvae ready to invade the pig.

female, and is very commonly absent from eggs which are removed from the uterus. This albuminous covering is stained a yellowish to a yellowish-brown by the coloring matter of the feces. The egg cell is usually in an unsegmented state at the time it is deposited. It almost completely fills the shell, and its nucleus is usually concealed by the large amount of coarse yolk granules.

Abnormal, unfertilized, and atypical fertilized eggs also occur in the feces of hosts and in the uteri of females. The unfertilized eggs are designated by their elongated ovoid forms (80 x 45 microns). The shell is very thin and smooth compared with the fertilized ones, and its contents consist

of refractive granules which completely fill the shell. The atypical forms vary in size and shape, some being spherical in shape while the major axis of others is greatly increased in length without the diameter increasing beyond that of an average egg, thus giving it a narrow, elliptical form instead of the broad oval which is more or less typical of the average egg. Similar observations were also reported by Foster (27), who made observations on a large number of *Ascaris* eggs and reported great variations as to the length of the eggs. This author observed a variation of as much as 51 microns between the shortest and longest egg. In one instance he found an egg that measured 107 microns in length with a diameter of only 39 microns.

INCUBATION UNDER NATURAL AND ARTIFICIAL CONDITIONS

Eggs deposited in the intestine of swine by the adult *Ascaris* are with very few exceptions in an unsegmented stage and segmentation rarely begins during their passage thru the intestine. Therefore, if the eggs are not promptly eliminated from the intestine of the host, development of the embryo is retarded. Martin (59) found by experimentation, with the pig *Ascaris* egg in vitro at 38° C., that segmentation is extremely slow and quite irregular. He found that about two-thirds of the eggs did not begin to divide, while the remaining third reached the two-blastomere stage. He also observed that if the ova were permitted to remain at this temperature for a short time they would die and degenerate. Thus, for development of the embryo the egg must be subjected to a temperature lower than that of the body. Because of the inhibitory effect of the body temperature it becomes necessary for the eggs to pass out of the intestine before they can develop to the infective stage. Development of *Ascaris* eggs to the vermiform stage outside of the animal body was first discovered by two Dutch investigators, Schubart and Verloren (in Leuckart, 51), in 1854. Richter (in Küchenmeister, 48) made the same observation independently in Germany the following year. Davaine (17), however, was the first to see the various stages of development of *Ascaris* eggs. Development of the eggs outside the animal's body is mainly influenced by three factors — temperature, moisture, and the supply of oxygen.

At low temperatures, development is very slow and if low enough may discontinue entirely; thus the time required for

full development of the embryo may be from a few days to months or even years; at very low temperatures the vitality becomes greatly reduced and in some instances the eggs are completely destroyed. It was observed by Leuckart (51) that embryonated eggs were not affected by being exposed to freezing for several weeks. Cram (14) noted that fresh eggs, subjected to a temperature of from 2° to 16° F. below zero for 40 days, partially developed eggs at the same temperature for 20 days, eggs containing fully developed embryos at a temperature of from 6° to 17° F. below zero for 10 days remained viable, and that motile embryos developed in the fresh and partially developed eggs when placed at a temperature of 24° C. The life of the embryos in the above eggs was found to be relatively short; for instance, the fresh eggs kept for 40 days at a temperature below zero contained very few motile embryos on the sixth day after motility was observed. The above-mentioned writer also observed that a group of eggs containing fully developed embryos were destroyed when they were exposed to a temperature of from 6° to 17° F. below zero for 20 days. Studies of the effect of low temperatures were also made by Bakker (5). The investigations of this author showed that the eggs of *Ascaris lumbricoides* were not affected when exposed to a temperature of — 15° C., but some of the eggs were killed when they were subjected to a temperature of — 20.5° C. and — 25° C., while those exposed to — 30° C. and below were destroyed. Bakker also observed that the eggs which survived low temperatures were retarded in their development. This delay was found to be increased by the lowering of the temperature and also by extending the time of exposure. The writer kept eggs at a temperature ranging from — 5° C. to 10° C. (the temperature was usually between 0° C. and 5° C.) in physiological salt solution for over four years, after which time the eggs were examined and found to be unchanged. The eggs still in an unsegmented condition were removed (March 23, 1926) from the above temperature and placed at room temperature (21° C. to 26° C.). After 14 days the eggs were found to contain motile vermiform embryos. These eggs, after being incubated at room temperature for a month, still contained many motile embryos. At this time a large number of the eggs were fed to cavias. Six days after the ingestion of the eggs these cavias were found to be dead. Upon autopsy the cause of death was found to be that of a severe *Ascaris* pneumonia.

Martin (59) observed the optimum temperature for the development of eggs from the pig *Ascaris* to be about 33° C. This investigator noted that it required from 9 to 13 days for the embryo to reach the rolled or coiled stage. Ransom and Foster (78) showed that a considerable number of the pig *Ascaris* eggs kept at this temperature contained fully developed embryos at the end of ten days, and that practically all reached complete development within a month. The writer, when he placed eggs at a temperature of from 31° C. to 34.5° C., found them developed to the two-cell stage after one day, the fourth day the mulberry stage was reached, and on the fifth day the mulberry stage was well advanced with one pole lighter in color, which according to Leuckart (51) developed into the head end. The seventh day a few moving embryos were noted and on the tenth day a few completely developed ones were observed in some of the eggs.

In the absence of moisture, development is inhibited, and extreme dryness may destroy the egg entirely. The moisture requirement, however, is very slight. Ross (81), many years ago in India, placed slides containing ova of the human *Ascaris* in the sun for six weeks, after which time they had become completely desiccated, but on examining them he noted that the embryos had fully developed and were living, and moving within the shell. Ransom and Foster (78) kept eggs of the pig *Ascaris* in an incubator at 37° C. until they became extremely dry, but there was no further development when they were moistened and exposed to a lower temperature. The author, after permitting a number of female pig *Ascaris* to become extremely dry, placed them at a temperature of from —5° C. to 10° C. (in a refrigerator) for a period of three years. Observations were made to determine the amount of evaporation that takes place when female *Ascaris* are subjected to the conditions described above, and found that they will dry to within approximately 5 per cent (4.92 per cent) of total desiccation by the end of two months.

After one year, an *Ascaris* was removed from this temperature and placed in distilled water for one day. The uterus, still in a dried condition, was removed and rubbed up in a mortar in order to force out the eggs. The eggs were then placed in an incubator at a temperature of from 31° C. to 34.5° C., and at the end of ten days fully developed embryos were noted. At the end of 25 months another *Ascaris* was removed from the refrigerator and treated the same as the dried female which was removed after one year, with the exception that the eggs were incubated at room temperature

(21° C. to 26° C.). These eggs were examined from time to time and at the end of 20 days a large number were found to contain fully developed embryos. The dried females which had remained in the refrigerator for three years were removed. The eggs contained in the uteri of these females were examined and found to be unsegmented, but they were somewhat distorted on account of their dried condition. These eggs were placed at a temperature of 31° C. and examined at various intervals for several months and during this time there was not the slightest evidence that segmentation ever took place. Therefore, the eggs contained within the uteri of these dried ascarids remained viable for a period of at least 25 months. Thoro dryness may prove fatal to the eggs of *Ascaris*, altho it is certain that under most climatic conditions there is sufficient moisture in the soil, or whereever the eggs may be after elimination from the host, to enable them to develop at least intermittently if not continuously.

Hallez (41) presented evidence in 1885 which showed that oxygen is necessary for the development of *Ascaris* eggs. His investigations showed that development was very rapid in eggs which were cultivated in humid air or oxygen, while the maximum slowness is attained in eggs incubated in boiled water, and in an atmosphere of carbon dioxide, hydrogen, and nitrogen. This investigator also found that development of the eggs ceases when they are deprived of oxygen, regardless of the stage of development, but they resume regular development when given oxygen. Wharton (107) introduced some eggs into water which had been boiled and covered by a film of oil to prevent the entrance of air. At the end of seven days none of the eggs had begun to develop; but upon removal to fresh water, development proceeded. Ransom and Foster (78) observed that if eggs are kept in a stoppered bottle filled with water, development is inhibited. Fauré-Fermiet (26) found that the determining factor of segmentation is oxidation of the hydrocarbons and fatty reserves stored up in the egg, a reaction which requires a supply of oxygen.

Ascaris eggs used for experimental purposes have been obtained and incubated in various ways by different investigators in order to secure rapid development of a high percentage of eggs to the infective stage. A number of the earlier workers used water as a medium (Richter (in Küchenmeister, 48), Leuckart (51), etc.), but Lutz (54) and Epstein (23) used an infusion prepared from feces and in most cases

the eggs were incubated at room temperature. Leuckart (51) noted that eggs developed more rapidly in moist earth than in water. Hallez (41) and Vajda (103) found glycerin to be a very satisfactory culture medium. Martin (59) observed that the pig *Ascaris* eggs developed very regularly when they were placed in a two per thousand solution of hydrochloric acid at 33° C. Wharton (107) obtained eggs for experimental purposes by taking living female ascarids from the intestines and placing them in Kronecker's solution (normal salt solution to which 0.06 grams of sodium hydroxide per liter is added). In this solution these parasites will remain alive for from 6 to 12 days, during which time they generally lay a large number of eggs. The eggs deposited were collected from the container in which the worms were kept, and placed on the surface of moist earth and in water at room temperature. This worker found that it required from 10 to 14 days for development of the eggs during the spring months. Ransom and Foster (78) collected eggs from large females. The uteri were dissected out and removed. These were then snipped into as small pieces as possible, and then rubbed up with a small quantity of 2 per cent formalin in a glass mortar to force the eggs out of the small uterine sections and break up the clumps of eggs. The eggs obtained by the above procedure from twenty uteri were evenly spread on the bottom of three Petri dishes 13.5 centimeters in diameter, and covered with a solution of 2 per cent formalin not exceeding one-fourth of an inch in depth. These cultures were then incubated at 33° C. to 34° C. At the end of 10 days, about one-third of the fertilized eggs contained motile, fully developed embryos, and practically all were fully developed by the end of a month. Baudet (7) collected eggs from the surface of glycerin-treated feces on filter paper. The filter paper containing the eggs was placed in Petri dishes for incubation. In one instance the filter paper was moistened with one per cent boric acid. Baudet also removed the eggs from the filter paper, and washed them free of glycerin before incubating in water. These investigations did not show any variation in the rapidity of the development of the eggs when incubated in the above media.

The author, in collecting and incubating eggs for experimental purposes, used the method of Ransom and Foster (78) with modifications. After the uteri were ground up in a mortar with five-tenths of one per cent formaldehyde solution, the suspension was passed thru a fine sieve to remove the large clumps of uterine material. The clumps were then

ground fine in the sieve by means of a pestle and in this way the maximum number of eggs was removed. The eggs, after being placed in Petri dishes 14 centimeters in diameter, were incubated at room temperature (21° C. to 26° C.). Cultures prepared from the entire uteri contain many incompletely developed eggs. This may be avoided to some extent by using the anterior portion of the uterus, and it is usually desirable to do so in order to keep the number of partially developed eggs in the culture reduced to a minimum.

THE LONGEVITY OF ASCARIS EGGS

It has been shown that *Ascaris* eggs will live for a long time, altho the maximum period that these eggs may remain viable is not definitely known. Richter (in Küchenmeister, 48) in 1885 found living embryos in eggs that had been in water for 11 months. Davaine (6) kept human *Ascaris* eggs for five years and at the end of this time two-thirds of the eggs remained intact. The eggs were administered to a rat and 12 hours later living larvae were found in the intestine. Leuckart (51) in 1857 reported that human *Ascaris* eggs may remain alive for two and one-half years. Epstein (23) in his experiments produced infections with human *Ascaris* eggs that had been kept in a culture of feces for one year. Morris (62), upon examining human feces that had been preserved in two per cent formalin, noted eggs which contained living embryos. Twenty-nine months later the eggs in the feces were still found to contain actively motile embryos. Ransom and Foster (78) state that they have kept pig *Ascaris* eggs alive for many months. Fülleborn (33) in some of his investigations noted living embryos in eggs preserved in formalin four to five years. The author saw living embryos in eggs of the pig *Ascaris* which were kept for 23 months at room temperature. This culture was again examined at 26 months, at which time the eggs were all found to be dead. Eggs were also kept alive at a low temperature for over four years, at which time they were removed to room temperature, where fully developed embryos were formed. These eggs were found to be alive two months (time of writing) after being placed at room temperature. It is apparent that the pig *Ascaris* eggs remain viable for a long time, and there is no reason to suspect that the eggs of this parasite retain their viability for a shorter period than those of the human *Ascaris*. From the above observations, it is evident that soil which is continually polluted with feces from man and pigs that are infested with *Ascaris* will in the course of time be

heavily seeded with living *Ascaris* eggs. It is also apparent that soil will contain viable eggs for a long time without being freshly contaminated.

THE EFFECT OF CHEMICALS ON THE ASCARIS EGGS

Ascaris eggs are extremely resistant to chemicals and are not killed by the ordinary disinfectants. The shells are impermeable and insoluble in many chemicals. Martin (59) found that hydrochloric acid in a two per thousand solution makes a very satisfactory culture medium. A 2 per cent solution of formaldehyde also gives the same opportunity for development, and eggs have been kept alive in this strength of formaldehyde for 29 months by Morris (62). The writer has also observed that the pig *Ascaris* eggs will survive for a period of 23 months in a 2 per cent formaldehyde solution. Ransom and Foster (78) state that a 10 per cent solution of potassium bichromate makes a suitable medium for growth. Many observations have been made upon *Ascaris* eggs with various chemicals that are highly destructive to protoplasm. Galli-Vallerio (34) was successful in developing *Ascaris* eggs to the vermiform stage in solutions of hydrochloric, sulphuric, nitric, and acetic acids which were from 2 to 50 per cent in strength. He also noted that eggs placed in saturated solutions of copper sulphate, iron sulphate, acetate of copper, and from 2 to 50 per cent of antiformin will develop embryos to a vermiform stage. Ransom and Foster (78) observed that full strength antiformin dissolved the shells of the eggs containing motile embryos, but the thin membranous lining remained intact. They showed that embryos within this membrane may still be active at the end of five days. Vajda (103) also exposed *Ascaris* eggs to antiformin and found that the outer capsules of the eggs were dissolved, while the inner membranes were only destroyed after death of the embryos.

Ransom and Foster (78) were able to keep embryonated pig *Ascaris* eggs alive for several hours in phenol. They also found embryos to be alive in eggs which were kept in crude petroleum for five weeks. There were living embryos noted in eggs that had been kept in petrolatum for ten weeks, but no embryos were found in eggs kept in crude petroleum for the same length of time.

The work of Cram (14) seems to show that an aqueous solution of 5 per cent phenol and 3 per cent cresol will prevent fresh and partially developed eggs from undergoing further development. The activity of embryos was also de-

stroyed in eggs exposed to those solutions. The destruction of these eggs was brought about by an exposure of ten hours in the phenol and five hours in the cresol solutions.

Yoshido (110) noted that embryos will develop in eggs kept in a 0.3 per cent solution of phenol, but that they were unable to develop in a 0.4 per cent. He also found that eggs will develop in a 12 per cent solution of formalin, and will live in a one per cent solution of corrosive sublimate. He also observed that eggs will undergo degeneration when they are exposed to human urine.

In view of the great resistance of *Ascaris* eggs to chemicals, it is evident that the ordinary methods of disinfection are of little avail in preventing the spread of *Ascaris* infection.

THE MODE OF ENTRANCE OF ASCARIS

The common avenue of infection with *Ascaris* under natural conditions is undoubtedly the swallowing of eggs containing fully developed embryos. The possibility of natural infection other than by the ingestion of eggs, however, cannot be entirely excluded.

Martin (59) injected embryonated eggs of *Ascaris vitulorum* and *Ascaris equorum* beneath the skin of a dog, rat, and cavias. These animals were killed from 9 to 41 days after the inoculation of the eggs. The cavia which was examined 9 days after being inoculated contained a large per cent of hatched eggs and eggs in the process of hatching at the point of inoculation. He also noted actively moving larvae in the pus. Another cavia examined 22 days after inoculation was found to contain larvae at the point of inoculation which had begun to change in form and grow. In a third cavia, he observed that all the eggs had hatched at the point of inoculation by the end of 41 days. In this animal the larvae were scattered in the pus, dead and degenerated, but these larvae did not seem to show any change in development.

Ransom anl Foster (78) repeated Martin's experiments by injecting infectious pig *Ascaris* eggs under the skin of three guinea pigs. Their experiments showed that hatching of *Ascaris* eggs will occur when introduced beneath the skin of cavias, and that larvae will appear in the lungs within a few days after inoculation, reaching the same stage of development that they would in a similar time if the eggs had been swallowed.

The author inoculated four pigs subcutaneously with *Ascaris* eggs (Experiment No. 13) and upon examination of one pig eight days later found many empty shells and free dead larvae at the point of inoculation. The writer also examined the lungs and other organs for evidence of migrating larvae, and not the slightest suggestion of migration was revealed.

A second pig was killed about one and one-half months after the inoculation and there seemed to be no evidence that hatching had occurred. The digestive tract of this animal was also found to be free of macroscopic ascarids.

Ascaris may also reach the small intestines of a definite host by means of a prenatal infection and by an intermediate or temporary host. The question of prenatal infection will be discussed in another chapter, and therefore need not be dealt with here.

Infection by means of an intermediate or temporary host was suggested by Stewart (87) in 1916. His observations led him to suspect that the transfer of larvae from the bronchi of rats and mice to the intestine of pig and man could be readily effected. He (88-94) later reported the feeding of infected lungs and caeca of rats and mice to pigs. The results obtained in these experiments were, however, rather inconclusive, altho one group of experiments probably suggests that pigs will develop adult worms from the ingestion of infected lungs.

The writer fed rabbit and calf lungs, which were heavily infected with *Ascaris* larvae, to five apparently *Ascaris*-free pigs. In this experiment (Experiment No. 9) the pigs were examined at different intervals after the feeding of the infected material, and the small intestines of the pigs were found to contain from seven to one hundred *Ascaris* of different sizes which, without doubt, originated from the feeding of the infected rabbit and calf lungs.

From the data set forth in Experiment No. 9, it is evident that a definitive host can become infected by the ingestion of an intermediate or temporary host. In nature the probability of infection by the ingestion of infected animal tissue is perhaps rather remote, but nevertheless, this possibility cannot be entirely eliminated.

The possibility of animals becoming infected with *Ascaris* under natural conditions other than by swallowing infectious eggs may be regarded as more or less accidental.

HATCHING OF EGGS

When infectious *Ascaris* eggs are swallowed, the majority of them pass thru the stomach unhatched. Laboublène (49) states that embryos will occasionally leave the eggs in the stomach, but that hatching occurs more commonly in the small intestines. Lutz (54) enclosed some *Ascaris* eggs in a piece of parchment paper and swallowed them. At the end of two and one-half hours the parchment paper, containing the eggs, was withdrawn from the stomach, at which time the eggs were examined and found to be intact. Martin (59) observed a few empty shells and five embryos in the stomach of mice fed experimentally. The egg shells were irregularly torn and the few embryos seen by him were dead and had undergone alterations. Martin believed that the few apparently hatched eggs observed by him in the stomachs of the experimental mice were eggs that had been crushed by the teeth of these animals in chewing the dry, hard bread on which the eggs were given. The writer also observed a few empty shells and free embryos in the stomach contents of a guinea pig which was experimentally fed pig *Ascaris* eggs two hours before the examination was made. In this case the writer is certain that the embryos were not released from the shells by the cavia crushing the eggs with its teeth, as the eggs were administered by means of a small pipette; yet it appears quite certain that the hatching of *Ascaris* eggs does not generally occur in the stomach.

Davaine (19) fed infectious human *Ascaris* eggs to a rat which was killed 12 hours later. In the small intestine he found free, living larvae. In another experimental rat, which was fed *Ascaris* eggs, living larvae and unhatched eggs were later observed in the feces. Davaine took some unsegmented and some ripe eggs and placed them in a small flask which he covered with linen. The flask was then introduced into the stomach of a dog. Two days later it was passed in the feces. The unsegmented eggs remained in the flask unchanged, the embryonated eggs were no longer noted, but a number of free embryos were found to be present in the flask. Davaine never detected *Ascaris* eggs hatching in the stomach, and therefore concluded that they must do so after reaching the intestine. Lutz (55) fed a parchment paper sack containing embryonated eggs to a dog. This sack after being voided in the feces was found to contain the eggs in an undigested state and four free larvae.

Davaine (19) noted that the action of the gastric juice was insufficient to dissolve the egg shells; he was also unable

to dissolve the shells by the intestinal juices. The experiments of DeKlug (47) reveal the fact that artificial gastric and tryptic digestion have no effect on the cell membrane of the *Ascaris* eggs. The work of Martin (59) also indicates that artificial and natural digestive juices do not dissolve the shells of the *Ascaris* eggs.

From the results obtained by Davaine (19) and Martin (59) it is evident that the hatching of the eggs is brought about by the activity of the embryos within the shell. The shell bursts and the embryo emerges thru the opening by its own efforts, a process which was observed by Laboublène (49) in the intestine of a rat. What factor or factors determine the hatching of the *Ascaris* egg has not as yet been determined. Davaine (19), after establishing the fact that eggs were not dissolved in gastric or intestinal juices, concluded that the intestinal juices, instead of dissolving the shell, soften it so that the embryos breaks thru it when stimulated to great activity by the warmth of the body of the host.

The results obtained from a considerable number of experiments in vitro with various media, permitted Martin (59) to reach the following conclusions as to the factors that determine hatching.

"Hatching of *Ascaris* eggs depends upon three factors. First, complete development of the embryo; second, a medium of an alkaline or neutral reaction; third, a temperature the same as that of the host of the parasite. The action of the digestive juices is not a diastatic one; they do not dissolve the shell in order to liberate the embryo. Hatching does not take place in the stomach because the medium is acid; it comes about in the intestine, because the medium is alkaline."

Ransom and Foster (78), in experiments with pig *Ascaris* eggs in vitro, were unable to cause hatching with any regularity, altho they did observe that a few eggs will hatch in vitro in almost any media, not only at body temperature, but at lower temperatures. The large majority, however, do not hatch outside of the animal body, even tho the eggs do contain embryos which are alive and active. From the above evidence it appears that the factors which influence the hatching of *Ascaris* eggs are as yet to be determined.

Altho ignorant of the factors that cause the hatching of *Ascaris* eggs, we are nevertheless able to reach certain conclusions as to the place and way in which hatching occurs. These may be summarized as follows: When *Ascaris* eggs containing fully developed embryos are swallowed, they pass into the stomach, where a few of them will hatch, but the

majority pass thru the stomach unhatched into the small intestine where they begin to hatch as early as two hours after they are ingested. The egg shells are not dissolved by the digestive juices, but the embryo is released by a V-shaped split in the shell thru which the embryo emerges by its own effort. *Ascaris* eggs do not hatch in the definitive host only, but in almost any mammal that ingests eggs which have reached the infective stage. It was pointed out by Danheim (16) that hatching of the *Ascaris* egg may also occur in birds.

THE LARVAL STAGE OF ASCARIS

DESCRIPTION OF THE LARVAE

There is considerable variation in size and structure of larvae from the time of hatching up until the time they reach the adult stage of development.

The larvae vary somewhat in size when hatched. They usually measure between 0.2 and 0.3 mm. in length, altho they may reach a length greater than 0.3 mm. Leuckart (51) in his description of newly hatched larvae states that they may attain a length of 0.38 mm. The diameter is more or less uniform thruout. The maximum diameter varies in different individuals from 0.013 mm. to 0.015 mm. On the anterior surface of the head a small rounded knob, the so-called "boring tooth," may be found. This knob or tooth, according to Leuckart (51), is nothing more than a thickening of the chitinous covering. In 1891 Stiles (100) described it as being composed of three parts resembling, by their position and form, the lips of the adult *Ascaris*. The oesophagus measures from about 0.078 to 0.09 mm. in length. In the living specimen its outline cannot ordinarily be distinguished except at the posterior end and the nerve ring cannot be seen. This portion and that of the body posterior to the intestine are very clear, and free from color or conspicuous granules. The distance of the excretory pore from the anterior end of the body is about 0.06 mm., and the distance from the anus to the tip of the conical tail is about 0.045 mm. The genital primordium is not manifest in the living specimen.

Previous to hatching, the larvae is enclosed in a close-fitting delicate, cuticular sheath. This sheath may be retained, or in some cases it may be cast off as it emerges from the shell. At the anterior end of the sheath there is a crown of minute papillae, there being apparently six in number.

Under normal conditions the presumption is that larvae molt at least twice; the first molt evidently comes about be-

fore hatching, at the time of hatching, or shortly thereafter; the latter molt apparently occurs before the larvae reach the stage at which they migrate from the lungs to the intestine.

The larvae in the liver usually undergo considerable change altho in a case where rapid migration takes place there may be very little change from those found at the time of hatching. The author has observed larvae in the liver varying in length from 0.197 to 0.6 mm.

In the lungs, the larvae vary in length from 0.228 to 2.4 mm. and from 0.014 to 0.1 mm. in maximum diameter; the length of the oesophagus is from 0.148 to 0.23 mm.; the distance of the nerve ring from the anterior end of the body is 0.08 mm.; the distance of the excretory pore from the anterior end of the body is 0.09 mm.; the distance of the anus from the tip of the tail is 0.054. The last three measurements were made on larvae originating from the lung of a cavia which died eight days after infection.

The general shape of the body is cylindrical, tapering slightly from the base of the oesophagus to the truncated head, and gradually from the junction of the middle and posterior thirds of the body, backward, and in the region of the anus the diameter has diminished approximately to half its maximum size. The tail is conical with its tip curved toward the dorsum. The cuticle is not ringed, and along each lateral line there is a well marked membrane, the lateral membrane, which curves toward the dorsum. The mouth is small, and the pharynx is very short. The oesophagus is club-shaped and is distinctly muscular, beginning almost immediately back of the mouth, and posteriorly projects into the anterior end of the intestine; the lips are not conspicuous; the knob-like process on the anterior aspect of the head which is characteristic of the newly-hatched larvae is no longer present. Immediately in front of the bulbous posterior end of the oesophagus, on the right or left side, is the nucleus of the cervical gland, which is about 0.018 mm. in diameter and contains numerous chromatin masses of varying sizes. The excretory pore is situated on the ventral line immediately behind the nerve ring. From it the excretory duct passes backward thru the group of cells of the ventral line. The intestine is cylindrical in shape and is composed of hexagonal cells and measures about 0.018 mm. in diameter. It extends from the posterior end of the oesophagus to the anal canal. The anal canal expands at the anterior extremity and is lined with a cuticle. The genital primordium is small, oval in shape, consisting of very few cells, and is situated on the

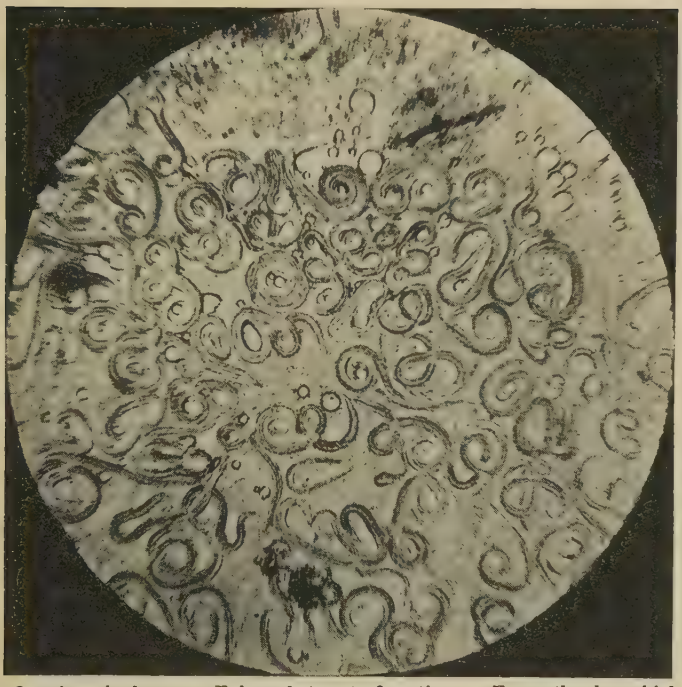


FIG. 3.— Ascaris larvae. Enlarged twenty-five times. From the bronchial mucus of a pig which died nine days after ingestion of infectious eggs.

ventral side of the intestine about the junction of the middle and posterior thirds of the body.

In the living larvae the oesophageal region and the region posterior to the anus are clear and transparent. The intestinal region is yellowish-brown thruout, due to the presence of numerous granules of this color in the intestine and intestinal cells.

The larvae in the intestine of the definitive host show development considerably in advance of those found in the lungs. The heads of 14-day-old larvae (2.5 to 3.8 mm. in length) found by Stewart (95-96) lost their larval appearance and had taken on the adult characteristics. The head bears three lips with four papillae characteristic of the adult. The internal surface of each lip is lined by thickened cuticle and is lined longitudinally. The cuticle of the body surface is marked by a series of rings.

The rudiment of the gonads is situated just behind the middle of the body length, and it consists of rudimentary

tubes leading to a rudimentary external aperture in the ventral line.

MIGRATION OF LARVAE IN THE BODY OF THE HOST

It has long been known that in artificially infected rats newly hatched *Ascaris* larvae may be found in the feces within a few hours after the eggs have been swallowed. Stewart (87), however, was the first to find that not all the newly hatched larvae pass out with the feces, but that some of the larvae will migrate out of the alimentary canal and reach other parts of the body. He found larvae in dilated blood capillaries in the liver of a mouse which died four days after being fed *Ascaris* eggs. The air vesicles of the lungs were also found to contain larvae in this animal. Ransom and Foster (78) have likewise shown by experiment that the larvae, after leaving the lumen of the intestine, will soon appear in the liver and may be demonstrated in this organ before they are evident in the lungs. They noted numerous larvae in the liver of a mouse which was killed 51 hours after feeding *Ascaris* eggs. In another mouse these investigators found larvae in the liver as late as eight days after feeding. Ransom and Cram (75) demonstrated larvae in the liver as early as 17 hours after ingestion of infectious eggs. Fülleborn (30, 33) has observed large numbers of *Ascaris* larvae 4 hours after feeding. The studies of Nettesheim (66) show that the larvae become numerous in the liver of mice 24 hours after the feeding of *Ascaris* eggs. The writer's experiments show that many were present in the liver of cavias as early as 15 hours after ingestion of *Ascaris* eggs. Several cavias were examined as early as four hours after ingestion of eggs, but no larvae were found to be present in the liver. Many larvae were also found to be present in the liver of a pig 21 hours after ingesting infectious *Ascaris* eggs.

Ascaris larvae become very numerous in the liver within 15 hours after ingestion of infectious eggs, and large numbers are still found to be present up to the fourth day. The larvae commonly disappear or at least become scarce after the fifth day, altho Ransom and Foster (78) have found numerous larvae in the liver of a mouse as late as 23 days after the ingestion of *Ascaris* eggs. The larvae reach the liver thru the portal circulation. Ransom and Cram (75) have noted large numbers of larvae in the portal vein as early as 17 hours after infection.

The path of migration from the intestine to the liver and thence to the lungs was first pointed out by Stewart (90).

He suggested that there are apparently two possible ways in which the larvae may travel from the intestine to the liver and later pass to the lungs. (a) The larvae, after boring thru the walls of the intestine, enter the mesenteric venules and are carried to the liver. In this organ they are arrested at the entrance of the hepatic capillary plexus. Extreme and acute fatty degeneration permits the penetration of the larvae along the capillaries between the columns of degenerated liver cells to the hepatic venules. The larvae then pass in the hepatic vein and vena cava to the right heart. From here they are carried to the lungs by the pulmonary artery. In the lungs they are arrested by the pulmonary capillaries. Embolism of the smaller branches of the pulmonary arteries occurs and is accompanied by hemorrhages of the arterioles and the larvae penetrate into the air vesicles and thence into the bronchi and trachea. (b) The larvae, after hatching in the intestine, migrate up the bile duct and reach the bile capillaries of the interlobular zone. They here work their way thru the degenerated liver tissues, reaching the hepatic venules, and continue their course as in the first case.

The first path of migration given was considered by Stewart as the more logical one.

Yoshido (109) reported results of experiments from which he concluded that the general and important course of migration of *Ascaris* larvae is thru the intestinal wall into the abdominal cavity, thence penetrating the diaphragm to enter the pleural cavity, and finally gaining access to the lungs from their surface. He further concluded that the migration of the larvae in the circulation, if it occurs at all, is merely accidental and of secondary importance.

Asada (3) also reports that his investigations with mice, rats, and guinea pigs show that *Ascaris* larvae will bore thru the intestinal wall, gain the abdominal cavity, wander toward the liver surface, and from here penetrate into the liver. From the liver the larvae gain the lungs by means of the blood vascular system. He also states that he was able to determine that the larvae migrate by means of the blood stream to the liver direct from the intestinal wall.

The observations made by Ransom and Foster (78) seem to be in accord with Stewart's first suggestion, that migration from the intestine to the liver may be by way of the portal system and from the heart to the lungs by way of the pulmonary arteries; altho they state that, on account of the difficulty of avoiding possible contamination with larvae from

other locations than the vessels from which the blood containing larvae was taken, the results of their experiments should not in themselves be accepted as sufficient proof of the migration of the larvae by the path indicated.

Ransom and Cram (75) in a very well organized series of experiments clearly demonstrated the passage of the larvae in the portal vein to the liver, thence by the hepatic veins and vena cava to the right heart, from which they reach the lungs by the pulmonary arteries.

The author found a larva in the right heart of a pig two days after the ingestion of eggs. This larva appears to be the first one reported found in the right heart of a pig.

Ransom and Cram (75) suggest further that larvae may reach the right heart by entering the lymphatic vessels in the intestinal wall and by being carried to the mesenteric lymphnodes, from here they pass in the lymphatic vessels to the receptaculum chyli, and thence by way of the thoracic duct pass to the right heart. They also examined the peritoneal and pleural cavities in a large number of animals and only rarely encountered larvae, and then in very small numbers, except in animals that died from 6 to 10 days after infection; in these as many as 35 larvae were found. The observations of Ransom and Cram certainly do not support the conclusions reached by Yoshido (109) that newly hatched larvae regularly penetrate the walls of the intestine, enter the abdominal cavity, and then burrow thru the diaphragm into the pleural cavity. The work reported by Fülleborn (28, 30, 33) is also opposed to the findings of Yoshido (109). He (30) states that the large majority of *Ascaris* larvae which escape from the eggs in the gastrointestinal tract reach the liver by way of the portal vein and not thru the peritoneal cavity. They migrate from the liver to the lungs by way of the hepatic vein and vena cava, and not thru the diaphragm. Fülleborn filled the entire thoracic cavity of a guinea pig with paraffine, after which a dose of infectious *Ascaris* eggs was administered. In this animal it was found that the lungs became just as heavily infected with larvae as did cavias which were not treated in this manner. Stewart (99) failed to find larvae in the pleural and peritoneal cavities in three cases during the first 48 hours after infection. The writer has also examined the peritoneal and thoracic cavities of 12 animals in from 18 to 169 hours after infection, with negative results in 10 instances (Experiment No. 4). One animal died 113 hours after infection with a very pronounced case of pneumonia and was found to contain one

larva in the thoracic cavity and one in the peritoneal cavity. The second animal, killed seven days after infection, was shown to contain two larvae in the pleural cavity. In the first case the animal was found to be dead, and disintegration of the tissue had taken place. In all probability the larvae escaped into the abdominal and pleural cavities after death. In the second case the larvae perhaps gained entrance into the pleural cavity thru defects in the pleura of the much hepatized lung. From the results obtained by the writer, Stewart (99), and Ransom and Cram (75), together with the conclusions reached by Fülleborn (30), it is evident that the principal path of migration of the *Ascaris* larvae from the intestine to the lungs is not thru the intestinal wall into the abdominal cavity, thence penetrating the diaphragm to enter the pleural cavity, and finally penetrating into the lungs from their surface, as concluded by Yoshido (109), but from the intestine to the liver by way of the portal circulation; from the liver they are carried to the heart by way of the hepatic vein and vena cava, from which organ they reach the lungs by the pulmonary arteries.

Stewart (87) noted larvae in the lungs of a mouse four days after it was fed *Ascaris* eggs. Ransom and Foster (78) have found larvae in the lungs of rabbits and cavias as early as three days after feeding *Ascaris* eggs. Ransom and Cram (75) have observed larvae as early as 17 hours after feeding eggs. The author has found many larvae in the lungs of a guinea pig as early as 15 hours after feeding. Fülleborn (30, 33) encountered a small number of larvae in the lungs as early as four hours after feeding eggs to guinea pigs. The author has examined a number of guinea pigs as early as 4 hours after after feeding infectious eggs, and noted a few hemorrhagic spots in the lungs but was unable to demonstrate the presence of larvae. In the lungs of cavias, rabbits, rats, and mice, larvae may be found rather commonly in large numbers from 4 to 10 days after ingestion of infectious eggs. The largest number are, as a rule, present between the fifth and the ninth day after feeding. As the larvae become numerous in the lungs, they become scarce or entirely disappear in the liver. The size of the larvae observed in the lungs by the writer varied in length from 0.228 to 2.4 mm. The larva which measured 2.4 mm. in length was found in the lung of a pig. Larvae measuring 1.8 mm. were observed in the lungs of a pig 8 days after infection.

Larvae have been observed to vary considerably in size when found in the same lung. The writer noted larvae

varying in length from 0.62 to 0.94 mm. in the lung of a cavia 7 days after infection. Ransom and Foster (78) reported even greater variations in size of larvae found in the lungs of numerous animals. These authors (78) have observed living larvae present in the lungs as late as 23 days after infestation and they have noted dead encapsulated larvae in the lungs of a rabbit as late as 86 days after infection. The writer observed many living larvae in the lungs of a calf 25 days after the ingestion of pig *Ascaris* eggs. Stewart (88) has found that larvae pass from the air vesicles of the lungs to the bronchi. He has seen larvae in the bronchi as early as the seventh day and in the trachea as early as the eighth day after infection. Ransom and Foster (78) detected a larva in the trachea of a rabbit 3 days and in a cavia 5 days after the ingestion of eggs. Nettesheim (66) observed larvae in the trachea of mice on the seventh day after infection. The writer has found larvae in the trachea of a cavia as early as 7 days, and in a pig as early as 9 days. Ransom and Foster (78) state that larvae are often found to be numerous in the trachea as early as the sixth day after infection. They have observed larvae as small as 0.23 mm. in length in a rabbit 3 days after infection. The writer has noted larvae which measured 1.15 mm. in length in a guinea pig 7 days after infection. One larva was detected as late as 21 days after infection in a guinea pig. There were many larvae present in the trachea of a calf as late as 25 days after ingestion of infectious eggs. From the trachea the larvae pass on into the larynx. The writer has found larvae in the larynx as early as 4 days and as late as 18 days after infection. The one noted 18 days after infection measured 1.66 mm. in length. Larvae, after having passed thru the larynx, arrive in the pharynx. The author recovered larvae from the pharynx of a pig on the tenth day. Stewart (88) observed larvae in the mouth of a mouse 8 days after infection. Koino (46) demonstrated one larva in his sputum 6 days after the ingestion of eggs, and on the eighth day he was able to demonstrate 178 larvae in the sputum.

From the pharnyx the larvae migrate down the oesophagus. Ransom and Foster (78) observed larvae in the oesophagus as early as 6 days and as late as 20 days (in a kid) after ingestion of infectious eggs. They found larvae in the oesophagus of a rabbit 8 days after infection which measured from 0.99 to 1.33 mm. in length. The writer has noted larvae in the oesophagus of a pig 10 days after infection. They

were also present in the oesophagus of a calf 14 days after infection.

Larvae, after migrating down the oesophagus, pass thru the stomach into the small intestine. The writer has found larvae in the stomach of a guinea pig as early as 7 days and as late as 11 days, and in a pig 10 days after infection. Ransom and Foster (78) have observed larvae, that had migrated thru the lungs, present in the small intestine of a guinea pig as early as 6 days after *Ascaris* eggs were fed. The writer has noted larvae measuring 1.15 mm. in length in the small intestine of a guinea pig as early as 7 days after ingestion of eggs. Larvae were also found in the small intestine of a pig as early as 9 days after feeding. Ransom and Foster (78) have encountered larvae in the small intestine of mice as late as 23 days after feeding. In laboratory animals such as rats, mice, cavias, and rabbits, the larvae continue their journey down the alimentary canal and pass into the large intestine. They can be found usually without difficulty in the caecum during this period. Stewart (89) has observed larvae in the large intestine of mice 9 to 12 days after they had been fed *Ascaris* eggs. He detected larvae in the feces of mice from the ninth to the twelfth day after infection. Stewart (94) also found dead larvae in the feces of a pig on the eleventh day after infection. Ransom and Foster (78) have demonstrated larvae in the caecum as early as 9 and as late as 23 days after infection. They have also observed larvae in the feces of a mouse as late as 13 days after infection which measured from 1.2 to 1.75 mm. in length.

In summarizing the observations that have been made on the migration of *Ascaris* larvae, it may be stated that: After hatching, the larvae burrow into the walls of the small intestine and enter either the venules or lymphatic vessels (when entering lymphatic vessels they are carried by way of the mesenteric lymphnodes), from there being carried to the liver, in the portal circulation, where they are distributed by the interlobular veins. They pass thru the capillary plexus between the interlobular veins and the central or intralobular veins, enter the latter and are carried to the hepatic veins. They may pass thru the liver rapidly or may be delayed for a time. Leaving the liver by the hepatic veins, the larvae are carried to the right heart in the vena cava. The larvae are carried from the right heart to the lungs by the pulmonary arteries where they are filtered out by the capillary network. In the lungs the larvae enter the

alveoli, and after further development and growth they pass on into the small intestine by way of the bronchi, trachea, larynx, pharynx, oesophagus, and stomach.

In rats, mice, guinea pigs, rabbits, and calves, *Ascaris* infection, as a rule, persists only two or three weeks, altho in the calf, according to Schwartz (84), the pig *Ascaris* may develop to sexual maturity. During the first few days after the eggs are ingested, there occurs an elimination of unhatched eggs and newly hatched larvae in the feces. About nine days after ingestion, the larvae which have completed the vasculo-pulmonary route begin to pass out of the body with the feces. They are, as a rule, practically all eliminated in less than three weeks after ingestion of the eggs. In the calf, however, they may be found to remain for a longer time. During their migration thru the body, they may increase in size from a length of 0.2 to 0.3 mm. to a length of 2.1 mm. [Stewart (89)]. Most of them, however, do not exceed 1.75 mm. in length, and some of them may be even less than 1 mm. in length when eliminated in the feces.

Some larvae evidently are not delayed in the lungs more than a short time, since they may be able to pass thru or around the capillaries in the lungs, return to the heart by the pulmonary veins, and be distributed to all parts of the body by the systemic circulation. Larvae have been found in aberrant organs which obviously they could not have reached unless brought there in the systemic circulation.

Stewart (87) has found larvae in the spleen between 4 and 6 days after feeding eggs. Ransom and Foster (78) observed them in the spleen in a number of cases. In a mouse, 13 days after infection, they noted larvae under the peritoneum in various places in the abdominal cavity, including the Fallopian tubes. Ransom and Cram (75) have frequently demonstrated the presence of larvae in the peripheral lymph nodes and in the thyroid and thymus glands. They have demonstrated larvae in the peripheral lymphnodes as early as 24 hours after infection. Fülleborn (29, 30, 32, and 33) has shown that, like *Strongyloides*, a number of *Ascaris* larvae attain the left heart from the lungs, and then are washed into all organs by means of the systemic circulation. He was able to demonstrate larvae in the carotid blood as well as in the brain capillaries, the kidneys, and other organs. The author examined the carotid blood in a number of guinea pigs and was not able to demonstrate larvae, but observed larvae in small subcapsular hemorrhages in a kidney of a pig 7 days after infection.

It is evident from the above observations that *Ascaris* larvae commonly reach the systemic circulation and are distributed to various parts of the body, apparently after passing thru the pulmonary capillaries and returning to the left heart by the pulmonary veins; tho Ransom and Cram (75) have suggested the possibility of larvae passing from the right to the left side of the heart thru an unclosed foramen ovale.

The infection route of the *Ascaris equorum* has also been shown, by Baudet (8), to be the same as that of the *Ascaris lumbricoides.*

DEVELOPMENT OF ASCARIS IN THE INTESTINE

Ascaris larvae, after having completed the vasculo-pulmonary circuit, migrate to the small intestine and, if in a definitive host (pig or man), settle down and develop to maturity. It is certain, however, that a number of larvae, even in a suitable host, after reaching the small intestine, are unable to establish themselves and therefore pass on out with the feces and perish, just the same as they do in case of mice, rats, guinea pigs, and rabbits.

Stewart (91, 94) has noted dead *Ascaris* larvae in the feces of pigs 11 days after infection. This would seem to be one of the probable explanations of the fact that pigs which have been fed *Ascaris* eggs experimentally and later show symptoms of lung migration by larvae are found to harbor only a few *Ascaris* or none at all, even tho they were not killed until after a lapse of time sufficient for the development of the parasite to maturity, or to a stage approaching maturity, in the intestine. This fact may be accounted for in other ways, one being that, in case of a heavy lung involvement, the larvae may succumb at this point and be perienterically digested. It may also be accounted for by the assumption that large numbers reach the digestive tract before they have undergone sufficient development, there to succumb and be digested. Such failures have been noted by Stewart (91, 94, 95), Ransom and Foster (78), and the writer. Stewart (91) fed six pigs infectious eggs, which resulted in a pneumonia; but upon examining the small intestines, he found five pigs to be free from intestinal *Ascaris.* In a second report (94), he experimented with six more pigs. In this experiment, three pigs showed evidence of pneumonia. The small intestines were examined about a month later, and it was observed that the small intestine of one pig contained 22 small *Ascaris,* which Stewart thought probably originated from the

experimental infection. He (95) later fed *Ascaris* eggs to two sucking pigs; pneumonia resulted 8 days after ingestion of the eggs. The small intestine of one of the pigs was examined 14 days later and was found to contain a large number of small *Ascaris,* which apparently originated from the feeding of the eggs. The second pig was killed 19 days later and was found to be entirely free of parasites. In another experiment he (99) fed four sucking pigs a large number of infectious *Ascaris* eggs. Serious symptoms of pneumonia were produced in these animals on the seventh day and one pig died. The remaining three pigs were examined 15, 17, and 19 days after ingestion of the eggs, and each pig contained many small larvae (3 to 7 mm. in length) in the small intestine. These larvae apparently originated from the feeding of infectious eggs.

In the foregoing reports of Stewart there seems to be a question as to whether the ascarids found in his experimental pigs originated from the feeding of infectious eggs, in that he does not present evidence to show that his experiments were controlled by pigs that were not given infectious eggs. Nevertheless, in the experiments in which he (95 and 99) reports the finding of small ascarids (2.5 to 7 mm. in length) in the small intestines of a number of experimental pigs, these ascarids in all probability originated from the feeding of infectious eggs.

Ransom and Foster (78) also experienced the fact that if pigs showing evidence of lung invasion are permitted to live long enough to enable the parasite to develop to maturity or to a stage approaching maturity in the small intestine, they will be found to harbor only a few *Ascaris* and in some cases none at all.

In one experiment they report the finding in the small intestine of eight immature *Ascaris,* which they suspect originated from the feeding of infectious eggs; but they do not consider the findings in this pig as conclusive evidence that the development of *Ascaris* in the intestine of swine follows the ingestion of eggs, nor that larvae that mature in the intestine first migrate to the lungs and back again before they settle down in the intestine.

In their report they state that there were possible sources of infection aside from the eggs that were fed, and this fact led them to state as follows:

"Owing to the difficulty of controlling experiments on pigs, we have not yet succeeded in obtaining sufficient evidence from experiments on pigs alone to demonstrate conclusively

that infection results from the ingestion of eggs and the subsequent migration of the larvae thru the lungs and back to the intestine, where they become established and develop to maturity."

The writer fed a number of pigs infectious *Ascaris* eggs, but noted that the *Ascaris* did not develop to maturity or a stage approaching maturity in the intestines, regardless of whether lung symptoms resulted or not. In other experiments, however, results were obtained which seemed to be quite conclusive. For example, in one experiment (Experiment No. 15), it was found that three pigs out of four harbored *Ascaris*. One of these pigs contained 73 approximately mature *Ascaris* in the small intestine. In the four controls used in this experiment, one pig harbored a single small *Ascaris*, while the intestines of the other three were found to be entirely free from this parasite.

The question raised by Stewart (87, 88) in regard to rats and mice acting as intermediate hosts induced the writer to conduct an experiment with pigs in which *Ascaris* infected lungs of rabbits and of a calf were fed to the pigs. In this experiment (Experiment No. 9) the five pigs which received the infected lung tissue were found to harbor from 7 to 100 mature or approximately mature *Ascaris* in the small intestines, while one of the two controls contained only three adult *Ascaris*, and the other none. From the results of this experiment it is quite evident that rodents can act as true temporary hosts and may occasionally serve as intermediate hosts under natural conditions, provided the rodents are not eaten before the larvae have developed to a certain stage; but it is also quite obvious that this method of infection is by no means a common one in nature.

Ransom and Foster (77) have shown by experiment that in lambs and kids the parasite will develop to a stage approaching maturity in the intestine following an infection brought about by the ingestion of pig *Ascaris* eggs.

Evidence has been presented which indicates that *Ascaris* larvae will also migrate thru the lungs in man. In 1888 Lutz (57) described symptoms in man which seem to indicate that the migration of *Ascaris* larvae occurred in this individual. An anthelmintic was later administered to this patient which caused 35 immature *Ascaris* to be eliminated in the feces. Koino (46) in a most courageous experiment upon himself was the first, however, to definitely demonstrate that the *Ascaris* larvae after having completed the vasculo-pulmonary circuit will, upon reaching the alimentary canal,

settle down and develop to the adult form. In this experiment he demonstrated the migration of the larvae up the trachea by collecting his sputum in which large numbers were found. Fifty days after the ingestion of infectious eggs, he took an anthelmintic which caused 667 immature *Ascaris* to be expelled. These parasites measured from three to eight centimeters in length.

From the evidence presented it is apparent that the *Ascaris lumbricoides* shows different degrees of adaptation in different animals. In such animals as rats, mice, cavias, and rabbits this parasite is able to undergo a certain portion of its development; that is, it reaches a stage in which it is ready to settle down in the small intestine, but is unable to develop to maturity and is digested and eliminated in the feces, altho some of the larvae are eliminated undigested. In case of the sheep and goat, the *Ascaris* apparently develop to a stage approaching maturity. Finally, in their definitive host, as man or pig, they are able not only to pass part of their cycle as is the case in imperfectly adapted hosts, but to continue their growth until they reach maturity.

According to the evidence at hand, the growth of *Ascaris* larvae, after their return to the small intestine, seems to be rather slow. Grassi (36) noted *Ascaris* eggs in the feces of a boy about two months after the feeding of eggs. In an experiment on children, Epstein (23) found the feces to contain large numbers of *Ascaris* eggs 86 days after the feeding of eggs. The feces were examined 12 days prior to the time of finding eggs; therefore it is evident that in these cases the parasites began producing eggs from the seventy-fourth to the eighty-sixth day after ingestion of ripe eggs; thus the time required for this parasite to reach full development in man appears to be about from two to two and one-half months after infection. Ransom and Foster (78) observed full-grown *Ascaris* in pigs 11 weeks old. The writer has found *Ascaris*, which contained fertile eggs, in pigs two months old. If Experiment No. 5 is valid, the writer was able to demonstrate *Ascaris* in a pig that contained fully developed eggs 49 days after feeding infectious eggs. It may, therefore, be concluded that the *Ascaris* in pigs may reach fertile maturity as early as 49 days after ingestion of eggs.

SYMPTOMS ASSOCIATED WITH MIGRATING LARVAE

The symptoms vary greatly depending upon the number of larvae migrating at one time. In animals in which only a

very small number of larvae migrate at one time, there may be no visible symptoms.

Animals in which a large number of larvae migrate simultaneously present more marked symptoms. The first symptoms noted are usually an elevation of temperature and evidence of pneumonia. The temperature may reach 106° F. in swine, but does not commonly remain at a high level for any great length of time — as is commonly seen in hog cholera and the pneumonias due to other causes — and usually disappears within four or five days. Respiration is increased up to 154 per minute and is jerky in character, the animal coughs frequently, anorexia is commonly noted with emaciation and occasionally the presence of diarrhea. There is generally an unthrifty appearance and interference with growth.

Ransom and Foster (78) were led to believe that a condition known as "thumps" was caused by the migration of *Ascaris* larvae, and stated that these losses "are undoubtedly often caused by *Ascaris* infection." The writer has examined a large number of these cases from the field and very little evidence to substantiate this opinion was encountered.

Under natural conditions very marked symptoms are rather uncommon in that the migration of large numbers of larvae simultaneously appears to be comparatively rare. The writer examined a large number of field cases for *Ascaris* larvae in the lungs, and in only a few cases did he find larvae in this organ, the largest number in a single case being three larvae. Of course, there is no doubt that heavy infections will occur occasionally. For example, Raffensperger (71) reported a field case of pulmonary ascariasis in which large numbers of migrating larvae were found in the lungs and bronchial secretions.

The losses by death are comparatively small, but the pigs that survive a severe *Ascaris* pneumonia may not entirely recover and fail to grow and develop at a normal rate. In addition to this, the pigs involved acquire an increased susceptibility to intercurrent infections.

Migration of *Ascaris* larvae may also produce symptoms in man. Mosler (63) reported a case of a child that died of worm fever. He also (in Leuckart 51) described symptoms of fever and dyspnoea in children which were observed several days after the administration of *Ascaris* eggs. In 1888 Lutz (57) reported that an experimental patient of his became sick with acid dyspepsia, frequent vomiting, and a slight remittent fever which was followed by an unusually

severe bronchitis. He suspected that this patient probably became sick from some unknown cause, but from our present knowledge it is evident that these symptoms were in all probability due to the migration of *Ascaris* larvae. Koino (46) in his recent experiments on his brother and himself with *Ascaris* eggs reports the occurrence of symptoms due to the migration of larvae. A few days after the ingestion of eggs, he observed a slight increase in temperature, which gradually became higher until it reached 104.3° F. The pulse was increased up to 120 per minute. It also became weak and thready. The respiration was increased and became shallow. During the height of the infection there was great difficulty in respiration and it was increased in frequency up to 58 per minute. There were other symptoms of pneumonia recorded. For example, there was dullness in various parts of the lungs. The râles and dullness increased until the crisis was reached; then the symptoms began to subside and on the thirteenth day the lungs were clear. Other symptoms, such as chills, severe headaches, flushed face, increased thirst, a decrease in the amount of urine, and heavy feeling over the chest, were noted. The patient coughed and the cough was increased in frequency with the increase in temperature. The cough came in paroxysms with intervals of two to five minutes at the height of the infection. The amount of sputum increased with the cough and was mixed with blood during part of the time; there were also varying numbers of larvae present and 178 larvae were found in 155 c.c. of sputum collected during a part of one day. The appetite was diminished from the very beginning and was entirely lost when the general symptoms became serious. There was a severe lumbago and pains in the gastrocnemius muscle. The olfactory nerve also became hypersensitive. The liver was enlarged and palpable.

Koino in his two experiments found that the symptoms were alike in both cases and that the seriousness of the migration depends upon the number of larvae migrating at one time.

LESIONS ASSOCIATED WITH THE MIGRATION OF LARVAE

Pathologic changes may be observed in almost any organ of the body as a result of migrating *Ascaris* larvae, tho the organs in which the changes are most manifest are the lungs, liver, and lymph glands.

The lesions in the lungs are more pronounced both macroscopically and microscopically than in any other organ. In

mild cases, there is very little alteration; there are small, bright red hemorrhages, varying in size from petechiae to ecchymoses, in which larvae can usually be demonstrated without much difficulty. The lungs are, as a rule, somewhat edematous. In more severe cases there are present many hemorrhagic areas varying in size from that of a pin point to several centimeters in diameter, giving the lungs a mottled appearance. There are also numerous small consolidated foci in the lungs. The bronchi and trachea often contain a frothy mucus, which is sometimes stained with blood. In extreme cases the lungs may be involved thruout, are greatly swollen, edematous, extremely hemorrhagic, dark reddish brown to a reddish black in color, and in some instances completely consolidated.

Microscopically the alterations in the lungs vary, depending upon the stage of migration and the number of larvae migrating at one time. In the early stages of the migration or when only a small number of larvae migrate simultaneously, the pathologic alterations are not very marked. In the beginning (28 hours after ingestion of infectious eggs) of the infection, one finds a slight, lobular pneumonia. There is a marked vascular congestion. The perivascular and peribronchial tissue is infiltrated with an inflammatory exudate. In the peribronchial tissue, there is a pronounced eosinophilia. Desquamation of the epithelium and numerous small hemorrhages were noted. At the acme of a heavy infection (five to ten days after the ingestion of infectious eggs), an extreme lobular pneumonia is noted. The exudate practically fills all parts of the lungs and it is composed of red blood cells, leucocytes, epithelium, numerous larvae, and some fibrin. There is a very marked eosinophilia thruout the lungs. The walls of the bronchi and the air sacs are greatly infiltrated with an inflammatory exudate. A marked vascular congestion was also noted. One likewise finds some proliferation of connective tissue, and as the pneumonia progresses the connective tissue becomes increased. Höppli (42), Cozinder (13), and Hadwin (39) have also noted a marked local eosinophilia in the lungs of experimental animals.

In the liver one finds an acute hepatitis within 48 hours after ingestion of infectious *Ascaris* eggs. The changes which take place in this organ are those of a marked fatty degeneration and extensive hyperleucocytosis. Many small inflammatory foci were scattered thruout the liver. A pronounced perivascular inflammation is also noted. One finds an eosinophilia thruout the liver tissue. It is especially con-

spicuous in the interstitium, and the perivascular tissue in this location is commonly very heavily infiltrated with oesinophiles. Numerous hemorrhages occur in the tissue. Vascular congestion and pigmentation thruout the liver are very common alterations. In some cases many giant cells are also found to be present. Occasionally larvae are noted during their migration thru the liver, in the dilated capillaries and free in the tissue. As the process progresses, one also finds a proliferation of the interstitial connective tissue. Joest (45) has described alterations in swine livers, designated as "hepatitis interstitialis parasitaria multiplex," which are very similar to those found in livers of pigs that had been experimentally infected with infectious *Ascaris* eggs. In all probability the lesions described by Joest resulted from the migration of *Ascaris* larvae; altho he indicated that he did not understand the nature of the parasite involved and suggested that consideration may be given to cestode or nematode larvae. Additional lesions have been described by other investigators. Nettesheim (66) observed small abscesses in the liver tissue, and occasionally these abscesses were found to contain larvae. Necrotic foci were also noted by Nettesheim (66), Cozinder (13), and Höppli (42) in the liver tissue. In the invasion of tissue by parasites, Hadwin (39) found that the eosinophiles may become reduced in the circulation, and that in case of severe migration of larvae the eosinophiles may entirely disappear from the circulation.

In man it is evident that the liver undergoes some changes during the migration of larvae, as Koino (46) has, by clinical methods, found the liver to be considerably enlarged.

The visceral and peripheral lymph glands are very commonly found to be edematous, congested, and enlarged. The groups of lymph glands that most commonly undergo alterations are the mesenteric, hepatic, and bronchial.

Other lesions occasionally found are those of an erythema, petechiae in the kidney (the number of petechiae varying from a few to many), gastritis, and enteritis. A slight hypertrophy of the heart was observed in one animal.

EXPERIMENTS ON THE LIFE HISTORY OF THE ASCARIS

The eggs used in the following experiments originated from *Ascaris* which were obtained from swine that had been slaughtered at the South Omaha packing plants and the Lincoln Packing Company plant. The eggs were removed from the large female *Ascaris* and incubated according to the author's method described in the chapter on incubation under natural and artificial conditions.

EXPERIMENT NO. 1

On April 4, 1921, four cavias were each fed a suspension of pig *Ascaris* eggs 51 days old.

The feces from the cavias were examined on the third, fourth, and fifth days after administering the eggs and in each instance empty shells and undeveloped eggs were found to be present.

On April 9, 1921, all four animals presented a rapid, jerky respiration, loss of appetite, and emaciation.

On April 11, 1921, two cavias were found to be dead. On autopsy one guinea pig was found to contain blood in the oral cavity and a bloody discharge from the nasal openings. Both pigs showed a very marked pneumonia. Larvae were noted in small numbers in the larynx, while in the trachea, bronchi, and lungs they were found to be present by the hundreds. In one of the animals, the stomach and small intestine each contained one larva. The larvae in the bronchi varied in length from 0.62 to 0.94 mm. and in diameter from 0.031 to 0.04 mm. The larvae observed in the intestine measured 1.15 mm. in length and 0.06 mm. in diameter. Some of the larvae in the trachea were also found to be as long as the one noted in the small intestine. There were no larvae present in the oesophagus, liver, spleen, heart blood, and thoracic cavity.

On April 12, 1921, the third cavia was found to be dead. On necropsy this animal also showed a very intensive pneumonia. The surface of the lungs was reddish-blue in color; the trachea and bronchi contained a thick mucus which contained many larvae. The lungs contained many larvae. A few larvae were also observed in the stomach and small intestine. No larvae were found in the larynx, liver, spleen, kidneys, heart blood, aorta, or rectum.

The fourth cavia was killed 11 days after it received the feeding of eggs. There was a slight pneumonia revealed by autopsy. A few larvae were noted in the larynx, trachea, bronchi, and stomach. The oesophagus, small intestine, spleen, and liver were free of larvae.

EXPERIMENT NO. 2

June 18, 1921. Two goats, three months old, were fed 10 c.c. each of *Ascaris* egg suspension which was 53 days old.

June 26, 1921. The appetite of both goats was reduced; there was slight dullness, coughing, and slight increase in respiration.

June 27, 1921. Goat No. 1 had a very pronounced pneumonia. Goat No. 2 was improved, but still coughed some.

June 28, 1921. The appetite of both animals was diminished. Goat No. 1 had a very rapid, jerky respiration, while the other goat showed only a slight abnormality in its respiration.

July 2, 1921. Both goats entirely recovered.

April 16, 1922. Goat No. 1 was examined by autopsy. The intestine

was examined for adult *Ascaris*, but none were found. Goat No. 2 was not examined by autopsy.

EXPERIMENT NO. 3

On February 16, 1922, four guinea pigs (Nos. 1, 2, 3, and 4) were fed *Ascaris* egg suspension which was incubated 142 days.

February 20, 1922. Guinea pigs Nos. 1 and 2 were killed and many larvae were noted in the lungs of both animals. There was also quite a pronounced pneumonia in both animals.

February 22, 1922. Guinea pig No. 3 was found to be dead and cavia No. 4 was killed. There was a bloody froth in the mouth and also the nasal openings. There was a pronounced pneumonia present and the lungs contained a large number of *Ascaris* larvae in both animals.

EXPERIMENT NO. 4

September 22, 1921, 4 P. M. Fed 20 guinea pigs 2 c.c. each of an egg suspension that was 149 days old. This experiment was conducted for the purpose of determining in part the path of migration and to what extent the larvae passed thru the liver.

Two cavias were killed each day for eight consecutive days; one animal was bled by cutting the throat and the other was chloroformed. The purpose of the carotid bleeding was to collect the blood and examine for larvae (all guinea pigs having uneven numbers were chloroformed).

The details of this experiment are presented in Table I.

In this experiment, larvae were found in the liver as early as 18 hours and as late as 113 hours after feeding. The maximum number (50) was noted in animal No. 7, 91 hours after the feeding of infectious *Ascaris* eggs.

In the lungs, the larvae were observed as early as 113 hours. In one instance (Cavia No. 10) there was one larva present in the peritoneal cavity and one in the thoracic cavity. In another case (cavia No. 13) two larvae were demonstrated in the thoracic cavity. In the first case the animal was found to be dead and disintegration of the tissues had taken place and in all probability the larvae escaped after death of the animal. The two larvae which were noted in the thoracic cavity of cavia No. 13 perhaps gained their entrance thru defects in the pleura of the very much hepatized lung. In no case was the writer able to find larvae in the heart blood, the carotid blood, or the blood taken from the portal vein and posterior vena cava.

EXPERIMENT NO. 5

Eight pigs were fed (by pipette) a suspension of *Ascaris* eggs on February 14, 1922.

April 5, 1922. The intestine of pig No. 1 was examined for *Ascaris*. Nineteen were found, of which 6 were large adults and the remainder small ones. Among the 6 large adults were 4 females reaching a length of 283 mm. and 4 mm. in diameter.

The 13 small *Ascaris* originated, in all probability, from the feeding of infectious eggs 49 days previous.

Ten of the small *Ascaris* were females. They varied in size from 125 to 189 mm. in length and from about 1.5 to 3 mm. in diameter. They were examined, and in each case it was noted that the uterus contained eggs which appeared to be fully developed and fertilized.

The uteri from the ones measuring 125 to 138 mm. were ground up to remove the eggs and incubated at room temperature in a two per cent formaldehyde solution. They were examined 15 days later (April 20, 1922) and the eggs were found to contain many moving embryos, which had almost reached complete development, indicating that the eggs were fully developed and fertilized, thus establishing the fact that these females were adults.

April 10, 1922. The intestines from pigs Nos. 2, 4, and 5 were examined.

In pig No. 2 there were four *Ascaris* found, of which one was a large female, two were small females, and the fourth was a male. The small females measured 150 and 151 mm. in length and 2.5 mm. in diameter. The uteri contained fertile eggs. The male measured 114 mm. in length and 2.5 mm. in diameter.

Pig No. 3 showed respiratory symptoms 7 days after feeding infectious eggs and was killed 2 days later. Upon autopsy a lobular pneumonia was noted, and the lungs were found to contain many larvae.

In pig No. 4 there were 10 *Ascaris* found, the smallest of which was a male and measured 168 mm. in length.

In pig No. 5 there were 56 adult *Ascaris* present, 20 of them males. The males measured 162 to 211 mm. in length and 3 to 3.5 mm. in diameter. The females measured 217 to 388 mm. in length and 3.5 to 4 mm. in diameter.

April 13, 1922. The intestine of pig No. 6 was examined for *Ascaris* and 21 found, of which 19 were females. One female was small, measuring 182 mm. in length and 3.5 mm. in diameter. The remaining females measured from 203 to 374 and the two males measured 185 and 199 mm. in length and 3 mm. in diameter.

Pig No. 7 was examined 74 days after the feeding of *Ascaris* eggs and found to be entirely free of *Ascaris*.

May 22, 1922. The intestine of pig No. 8 was found to contain 6 adult *Ascaris*, of which 2 were males. The males measured 211 mm. in length and the females had attained a length from 262 to 288 mm.

The evidence presented in this experiment does not permit definite conclusions in that it was not controlled by pigs which were not fed infectious eggs.

EXPERIMENT NO. 6

March 20, 1922, 6 P. M. One cavia was fed a 41-day-old *Ascaris* egg suspension. This cavia was killed 15 hours after ingestion of the infectious *Ascaris* eggs. Blood was drawn from the right heart, posterior vena cava, and portal vein. This blood was examined and no larvae were noted altho the lungs and liver contained many larvae. The ones in the lungs measured 228 microns in length and the ones in the liver measured 197 microns in length.

EXPERIMENT NO. 7

March 9, 1923. Three cavias (Nos. 1, 2, and 3) were fed a suspension of *Ascaris* eggs.

Cavia No. 1 was killed and examined one hour after the ingestion of eggs. The stomach and 28 centimeters of the small intestine were found to contain many unhatched eggs, of which a large number con-

TABLE I (Experiment No. 4)

Cavia No.	Examined after feeding *Hours*	Results								Remarks
				Blood				Cavity		
		Liver	Lungs	Heart	Carotid	Portal vein	P. vena cava	Pleural	Peritoneal	
1	9-23-'21 18 hours	7 larvae present	Neg.	Neg.	Neg.	Neg.	Neg.	Neg.	Neg.	Eggs were found in the stomach and small intestines.
2	"	Neg.	Neg.	—	—	—	—	—	—	
3	9-24-'21 44 hours	Neg.	—	—	Neg.	—	—	Neg.	Neg.	
4	"	Larvae present	—	—	—	Neg.	—	Neg.	Neg.	
5	9-25-'21 68 hours	11 larvae present	—	—	Neg.	—	—	—	—	
6	"	6 larvae present	—	—	—	—	—	Neg.	Neg.	
7	9-26-'21	50 larvae present	—	—	Neg.	—	—	Neg.	Neg.	
8	"	Neg.	—	—	—	—	—	Neg.	Neg.	
9	9-27-'21 114 hours	Neg.	—	—	Neg.	—	—	Neg.	Neg.	

TABLE I (Experiment No. 4)—(Continued)

Cavia No.	Examined after feeding *Hours*	Results								Remarks
				Blood				Cavity		
		Liver	Lungs	Heart	Carotid	Portal vein	P. vena cava	Pleural	Peritoneal	
10	9-27-'21 113 hours	5 larvae present	Many larvae present	—	—	—	—	1 larva present	1 larva present	Died of *Ascaris* pneumonia. Some post-mortem decomposition.
11	9-28-'21 137 hours	Neg.	—	—	Neg.	—	—	Neg.	Neg.	Sick with *Ascaris* pneumonia at time of killing.
12	9-28-'21 140 hours	Neg.	—	—	—	—	—	Neg.	Neg.	Sick with *Ascaris* pneumonia at time of killing.
13	9-29-'21 169 hours	Neg.	—	—	Neg.	—	—	2 larvae present	Neg.	Breaks in the pleura of the greatly hepatized lungs.
14	"	Neg.	—	—	—	—	—	Neg.	Neg.	Sick with *Ascaris* pneumonia at time of killing.
15	9-30-'21 192 hours	Neg.	—	—	Neg.	—	—	—	—	Sick with *Ascaris* pneumonia at time of killing.
16	"	Neg.	—	—	—	—	—	—	—	Sick with *Ascaris* pneumonia at time of killing.
17-18 19-20	—	—	—	—	—	—	—	—	—	These animals recovered from *Ascaris* pneumonia and were not examined.

The dash (—) in this table indicates that the material was not examined.

tained moving embryos. There were no empty shells or free larvae observed in these organs. The blood from the portal vein, vena cava, and right heart was examined and found to be free of larvae.

Cavia No. 2 was killed and examined two hours after feeding eggs. The stomach and small intestine contained a few hatched, many unhatched eggs, and a few living embryos.

Cavia No. 3 was killed and examined four hours after the ingestion of infectious eggs. The small intestine contained many hatched eggs. The blood from the portal vein, vena cava, and right heart was found to be free of larvae. A number of small pin point hemorrhages were observed in the intestinal mucosa.

March 17, 1923. Two cavias (Nos. 4 and 5) were fed a suspension of infectious *Ascaris* eggs.

Cavia No. 4 was killed and examined 19 hours after the feeding of eggs. The mesenteric lymph glands were found to contain *Ascaris* larvae. No larvae were noted in the blood of the portal vein, vena cava, and right heart.

Cavia No. 5 was killed and examined 4 hours after the feeding of eggs. No larvae were found in the portal vein, vena cava, and right heart. Small hemorrhagic areas present in the lungs were also found to be free of larvae.

EXPERIMENT NO. 8

In this experiment three calves (Nos. 1, 2, and 3) were fed a suspension of pig *Ascaris* eggs.

Calf No. 1 was 37 days of age when the *Ascaris* eggs were administered (April 11, 1923). This animal showed a slight rise in temperature 6 days after it received the dose of infectious *Ascaris* eggs. On the ninth day, there was an increased (93 per minute), labored, jerky respiration, and complete loss of appetite. The animal groaned with each respiration; there was also a cough and respiratory râles. A mucous discharge from the nasal opening and slight salivation was observed. On the tenth day, there was evidence of emaciation and the respiration had decreased in frequency.

Death of this animal occurred 14 days after the administration of the *Ascaris* eggs. The autopsy findings were those of a very pronounced lobular pneumonia and emphysema. There was a bloody mucus present in the nasal openings, larynx, trachea, and bronchi. The bronchial lymphnodes were also congested. Many larvae were found to be present in the lungs, bronchi, and trachea. Larvae were also present in the larynx, oesophagus, and small intestine in small numbers.

Calf No. 2 received the suspension of pig *Ascaris* eggs at 14 days of age.

This animal showed a marked increase in respiration (110 per minute) 9 days after ingestion of the infectious eggs. On the tenth day there was difficulty in breathing, the animal coughed, and groaned with each respiration. This animal was found to be dead 13 days after receiving the dose of infectious eggs. On autopsy a frothy mucus exuding from the nasal opening and pneumonia were noted. There were large numbers of larvae present in the trachea and all parts of the lungs.

Calf No. 3 at 34 days of age received a suspension of pig *Ascaris* eggs which had been incubated at room temperature for 7 months. Respiratory distress was noted 8 days after the administration of the

infectious eggs. Respiration became much increased and on the tenth day it had reached a frequency of 137 per minute. Respiratory symptoms of varying degrees were noted from the eighth day after the ingestion of the infectious eggs until the death of the animal which resulted 25 days after the feeding of the pig *Ascaris* eggs.

The autopsy findings were those of an intense pneumonia and an enteritis of the small intestine. There were many larvae found in the trachea. Large numbers of larvae and empty skins were also noted in the lungs; these were probably a result of the moulting of the larvae during their migration. The larvae in the lungs had reached a length of 2.12 mm. by 0.09 mm. in diameter. The intestinal tract was found to be free of larvae of macroscopic size.

EXPERIMENT NO. 9

In this experiment 30 rabbits and 7 pigs were used. The 30 rabbits were each fed a suspension of infectious pig *Ascaris* eggs on June 15, 1923. These rabbits showed respiratory symptoms 6 days after ingestion of infectious eggs. On the seventh day 15 of the animals were found to be dead, 13 died the following day, and the 2 remaining rabbits died on the ninth day. On autopsy all of the above rabbits showed a very extensive pneumonia. The lungs and trachea of each animal were found to be heavily infested with *Ascaris* larvae.

These 30 rabbits were kept in a pen with a number of other rabbits from the time they were fed the *Ascaris* egg suspension until death occurred. During this time, the rabbits which had been fed *Ascaris* eggs apparently contaminated the food and drinking water with infectious eggs, because 10 days after the 30 rabbits received the feeding of eggs 2 of the other rabbits were found to be dead. Both rabbits were carefully autopsied and it was found that death was due to a very extensive pneumonia in each case. The lung tissue of these animals, upon microscopic examination, revealed the presence of many *Ascaris* larvae, thus indicating that these rabbits were infected as a result of being in contact with the 30 rabbits which were artificially infected with *Ascaris* eggs.

June 22, 23, and 24, 1923. The respiratory organs of the 30 rabbits dead with *Ascaris* pneumonia were divided into five equal parts and fed to five pigs (Nos. 1, 2, 3, 4, and 5), which had been treated a number of times with oil of chenopodium and castor oil. The feces were also examined for *Ascaris* eggs before and after administration of the vermifuge, and at no time were eggs found to be present in the feces.

Pigs Nos. 6 and 7 which acted as controls were also added to the experiment, after they were found by fecal examination to be apparently free of *Ascaris*.

July 9, 1923. Pigs Nos. 1, 2, 3, 4, and 5 were each fed a portion of calf lung which was heavily infested with pig *Ascaris* larvae.

August 1, 1923. Pig No. 1 was found to be dead. This animal was carefully autopsied and the small intestine contained 52 small ascarids varying in length from 25 to 90 mm. and in diameter from 0.5 to 1.5 mm.

September 12, 1923. Pig No. 2 was killed and the entire intestinal tract was examined for *Ascaris*. This examination revealed that 12 *Ascaris*, 3 of which were males, were present in the small intestine. The females were found to measure 100, 120, 155, 162, 172, 182, 190,

and 195 mm. in length. They varied in diameter from 2.0 to 4.5 mm. The males measured 102, 120, 131 mm. in length and the smallest male measured 2.5 mm. in diameter.

September 19, 1923. Pigs Nos. 3 and 4 were killed and in each case the intestinal tract was examined for *Ascaris*.

In pig No. 3 the small intestine was found to contain 7 *Ascaris*, of which 2 were males. The females measured 115, 222, 230, 240, and 250 mm., and the males measured 170 and 187 mm. in length.

In pig No. 4 the small intestine was found to contain 100 *Ascaris*, of which 33 were males. The females measured from 160 to 255 mm. in length, the larger percentage measuring within 15 mm. of the largest one. The diameter varied from 3.0 to 4.5 mm. The males measured from 142 to 165 mm. in length. The diameter varied from 2.5 to 3.0 mm.

September 21, 1923. Pigs Nos. 5, 6, and 7 were killed and the intestinal tract of each animal was examined for *Ascaris*.

In pig No. 5 the small intestine contained 17 *Ascaris*, 12 of which proved to be males. The females measured 100, 215, 225, 230, and 260 mm. in length. The diameter varied from 1.5 to 4.0 mm. The males measured 125, 130, 130, 140, 143, 150, 160, 160, 167, 173, 175, and 175 mm. in length. They varied from 2.0 to 2.5 mm. in diameter.

Pig No. 6, a control animal, was noted to be entirely free of *Ascaris* in the intestinal tract.

Pig No. 7, a control animal, was found to contain 3 *Ascaris* in the small intestine, one being a male. The females measured 225 and 240 mm. in length and 4 mm. in diameter. The male measured 150 mm. in length and 2.5 mm. in diameter.

EXPERIMENT NO. 10

In this experiment, five 4-months-old pigs which were known to be free of *Ascaris* were used. Three of these pigs were given a definite number of infectious pig *Ascaris* eggs in a gelatin capsule; the 2 remaining animals were held as controls.

December 29, 1924. Pig No. 1 received 1,015 embryonated eggs, pig No. 2 received 1,047 embryonated eggs, and pig No. 3 received 2,149 embryonated eggs. Pigs Nos. 4 and 5 were held as controls and therefore were not given *Ascaris* eggs.

February 26, 1925. The above 5 pigs were killed and the stomachs and intestinal tracts were examined for adult *Ascaris*, and in each case these organs were found to be free of this parasite.

EXPERIMENT NO. 11

In this experiment a 25-year-old horse was given two large doses of infectious pig *Ascaris* eggs. This animal was kept under observation for 27 days after the first dose of eggs and 13 days after the second dose had been given. During this period the horse did not show the slightest evidence of larval migration. This horse was killed 27 days after the first and 13 days after the second dose of *Ascaris* eggs was given. A careful autopsy was made on this animal and there were no macroscopic ascarids found in the intestinal tract. The lungs appeared normal on gross examination, and no larvae were found in this organ microscopically.

EXPERIMENT NO. 12

March 16, 1925. Four 3-day-old pigs (Nos. 1, 2, 3, and 4) were each fed a large dose of infectious pig *Ascaris* eggs. A fifth animal, the same age, was used as a control.

Pig No. 1 was found to be dead 21 hours after it received the feeding of infectious eggs. This animal was carefully examined for larvae. The liver was noted to contain many larvae, while examination of the lungs and the blood of the portal vein, posterior vena cava, and right heart resulted in negative findings. A few eggs were present in the small intestine. In the large intestine very many unhatched, embryonated eggs and free larvae were observed.

March 18, 1925. Pig No. 2 was killed. No larvae were demonstrated in the posterior vena cava, portal vein, parts of the liver and the lungs. One larva was found to be present in the blood of the right ventricle of the heart. The large intestine contained many unhatched eggs and free larvae. In the small intestine a few undeveloped eggs were noted.

Seven days after the feeding of the infectious eggs, pigs Nos. 3 and 4 began to manifest lung symptoms and on the eighth day the pneumonia symptoms had become very pronounced. The respiration had become very rapid and jerky in character.

March 25, 1923, pig No. 3 was found to be dead. This animal showed a very extensive pneumonia on autopsy. The lungs and trachea were swarming with larvae. Many larvae, some of which had obtained a length of 1.5 mm., were noted in the small intestine. Larvae were also present in the stomach and large intestine.

May 25, 1925, pigs Nos. 4 and 5 were killed. The stomachs and small and large intestines of both animals were examined. In pig No. 4 these organs were noted to be entirely free of macroscopic *Ascaris*. The lungs of this animal were also found to be perfectly normal.

Pig No. 5 contained 2 small *Ascaris* (40 and 52 mm. in length) in the small intestine. This animal was kept in the pen with the 4 pigs which received the infectious *Ascaris* eggs and apparently ingested some of the eggs eliminated by these 4 animals.

EXPERIMENT NO 13

In this experiment four 15-day-old pigs were inoculated subcutaneously with a large dose of infectious pig *Ascaris* eggs.

April 7, 1925, pigs Nos. 1 and 2 received the infectious eggs subcutaneously in the axillary region and pigs Nos. 3 and 4 had the eggs introduced under the skin in the inner part of the thigh.

April 15, 1925, pig No. 1 was killed and a careful autopsy was made. At the point of inoculation large masses of unhatched eggs were found to be present. Some of these eggs contained fully developed embryos. The embryos showed a marked fatty degeneration. There were also many hatched larvae present (0.243 mm. in length) which were dead and had undergone degeneration similar to the embryos within the egg shell. Some of the hatched larvae were also completely digested. There were also empty shells, bacteria, and degenerated leucocytes at the point of inoculation. This material was entirely surrounded by a capsule made up of connective tissue. The lungs, trachea, bronchi, prepectoral lymphnodes, and the intestinal tract were found to be free of larvae.

May 19, 1925, pig No. 3 was killed. This animal contained an encapsulated mass about 7 mm. in diameter at the point of inoculation (inner part of the thigh). This mass consisted of large numbers of unhatched eggs, some of which contained fully developed embryos, bacteria, and cell debris. In this animal there seemed to be no evidence that hatching of the eggs had occurred at the point of inoculation, in that there were no empty shells or free larvae present. The shells of the un-

hatched eggs showed no alteration of any kind in that all parts of the shell seemed to be intact. The stomach and small and large intestines were found to be free of *Ascaris*.

Pigs Nos. 2 and 4 did not show any symptomatic evidence of larval migration and were therefore not autopsied.

The writer conducted other experiments on the life history of this parasite. The results obtained were similar to those described in the foregoing experiments and for that reason the writer felt it unnecessary to include them in this publication.

PRENATAL INFECTION WITH ASCARIS

The question of intrauterine infection with parasites has from time to time been discussed by a number of parasitologists in various parts of the world. Apparently the first reports presented in regard to this subject were those of two Japanese investigators. Fujinami and Nakamura (in Cort, 12) in 1911 reported the finding of the *Schistosoma japonicum* in a dog fetus. In 1914 Narabajashi (in Cort, 12) also noted the *Schistosoma* in 5 out of 7 embryos from an artificially infected dog. This author (in Cort, 12) later (1916) referred to the finding of 14 young specimens of this parasite in the placenta of two pregnant cavias that had been infected experimentally. In this publication he also mentions that the feces of 3 out of 22 newborn children were found to contain the eggs of *S. japonicum*.

There were also a number of reports published with reference to the possibility of intrauterine infections with several different species of nematodes. Neveu-Lemaire (68) noted *Dictyacaulus filaria* adults in the trachea of a 4-day-old lamb which he suspected of being a congenital infection. Howard (43) observed the presence of hookworm ova in the feces of a 14-day-old child. Alder and Clark (2) examined 13 young dogs (from 2 to 15 days of age), representing eight different litters, for *Anchylostoma caninum* and found 6 of them to harbor this parasite in various stages of development. Alder and Clark state that where hookworm infestation in dogs is intense, intrauterine infections appear to be common. Ackert and Payne (1) also reported the presence of mature *Necator suillus* in several 26-day-old pigs and indicate that it is possible that these pigs were victims of a prenatal infection.

Evidence was presented by Fülleborn (28-30-31-32-33) and Shillinger and Cram (85) which seems to indicate that pups may become infected in utero with *Balascaris* larvae. Fülleborn (30) found *Balascaris* two to three weeks old in a pup

four or five days after birth. He (31) later indicated that he was able to demonstrate intrauterine infection by injecting thousands of *Balascaris* larvae, obtained from the liver of an infected cavia, under the skin of a bitch. This bitch, 11 days after the inoculation of the larvae, whelped pups that contained hundreds of larvae. He noted larvae in the lungs and liver immediately after birth, and two days later larvae were observed in the intestine which measured 1 to 1½ mm. These observations induced Fülleborn to state that the *Ascaris* larvae reach the fetus by means of the placental circulation. Shillinger and Cram (85) administered two large doses of ripe *Balascaris* eggs to a bitch in an advanced stage of pregnancy. Eight days after the first and 6 days after the last feeding, 12 pups were born, 8 of which were dead at birth or shortly thereafter and the remaining 4 of which died the following day. They prepared pressed sections from fresh tissue of the liver, spleen, kidney, and lungs and found that 8 of the 12 pups showed *Balascaris* larvae. These findings led Shillinger and Cram to believe that the larvae undoubtedly reached the placenta thru the maternal blood stream. They also indicate that it is evident that all the larvae present in the pups resulted from the feeding of embryonated eggs.

Bollenger (9), Macfie (58), and Griffiths (38) reported the presence of *Ascaris vitulorum* in young calves which they suspected was the result of an intrauterine infection.

The various observations and experiments reported induced the writer to undertake an experiment with pregnant sows to determine whether or not intrauterine infection is a common occurrence in swine with the *Ascaris lumbricoides.*

This experiment was conducted on six young sows which were pregnant for the first time.

Four of these sows were fed from five to nine large doses of pig *Ascaris* eggs during the latter part of the gestation period. The number of eggs given to each sow probably amounted to several million. The two remaining sows were held as controls.

The eggs used in this experiment were tested out for infectivity on cavias. These eggs, when administered to guinea pigs, produced a fatal *Ascaris* pneumonia.

The pigs were examined at varying periods after birth. Some of the pigs were autopsied at birth while others were permitted to live for a considerably longer time. The results of this experiment are shown in Tables 2, 3, 4, 5, 6, 7.

TABLE II (Experiment No. 14)

Sow No. 1

Date of birth	Bred	Date farrowed	No. of pigs in litter	Remarks
3-16-'24	11-27-'24	3-21-'25	7	This sow was fed 5 large doses of infectious *Ascaris* eggs on the following dates: 2-17-'25; 2-19-'25; 2-21-'25; 2-24-'25; 3-12-'25.

Pigs from Sow No. 1

No. of pig	Date of death and autopsy	Results	Remarks
1	3-22-'25	Negative	This pig was found to be dead 22 hours after birth. The lungs, liver, spleen, stomach, and small and large intestines were examined for *Ascaris*.
2	3-25-'25	"	The lungs, liver, stomach, and small intestine were examined for *Ascaris*.
3	3-27-'25	"	The spleen, liver, stomach, and small intestine were examined for *Ascaris*.
4	4-17-'25	"	The intestinal tract was examined for *Ascaris*.
5	4-20-'25	"	The intestinal tract and lungs were examined for *Ascaris*.
6	4-23-'25	"	The intestinal tract and lungs were examined for *Ascaris*.
7	5- 1-'25	"	The stomach and intestinal tract were examined for *Ascaris*.

From the details recorded therein it will be observed that the four sows which received large quantities of infectious pig *Ascaris* eggs during the latter part of pregnancy farrowed 35 pigs. Of these pigs one was born dead, and the remaining 34 were all born in a good, vigorous state of health.

It will be noted that 21 of these pigs received consideration. Twelve of these animals were carefully autopsied at different ages and in each instance the examination yielded negative results. The nine remaining animals were each given an anthelmintic at 53 days of age, which yielded negative results.

TABLE III (Experiment No. 14)

Sow No. 2

Date of birth	Bred	Date farrowed	No. of pigs in litter	Remarks
1-12-'24	11-27-'25	3-23-'25	8	This sow was fed 5 large doses of infectious *Ascaris* eggs on the following dates: 2-17-'25; 2-19-'25; 2-21-'25; 2-24-'25; 3-12-'25.

Pigs from Sow No. 2

No. of pig	Date killed and autopsy	Results	Remarks
1	4-15-'25	Negative	The lungs, trachea, bronchi, and intestinal tract were examined for *Ascaris*.
2	5-19-'25	"	The stomach and small and large intestines were examined for *Ascaris*.
3-4-5 6-7-8	—	—	These pigs were not examined for *Ascaris*.

TABLE IV (Experiment No. 14)

Sow No. 3

Date of birth	Bred	Date farrowed	No. of pigs in litter	Remarks
3-30-'24	12-19-'25	3-31-'25	10	This sow was fed 9 large doses of infectious *Ascaris* eggs on the following dates: 2-17-'25; 2-19-'25; 2-21-'25; 2-24-'25; 3-20-'25; 3-21-'25; 3-23-'25; 3-25-'25; 3-27-'25.

Pigs from Sow No. 3

No. of pig	Date of autopsy	Results	Remarks
1	4-1-'25	Negative	This pig was killed 15 hours after birth and the lungs, stomach, and small intestine were examined for *Ascaris*.
2	4-3-'25	"	This pig was found to be dead 3 days after birth and the lungs, liver, spleen, trachea, stomach, and small intestine were examined for *Ascaris*.
—	—	—	The remaining 8 pigs were not examined for *Ascaris*.

TABLE V (Experiment No. 14)

Sow No. 4

Date of birth	Bred	Date farrowed	No. of pigs in litter	Remarks
3-11-'24	12-10-'24	4-3-'25	10	This sow was fed 9 very large doses of infectious *Ascaris* eggs on the following dates: 2-17-'25; 2-19-'25; 2-21-'25; 2-24-'25; 3-20-'25; 3-21-'25; 3-23-'25; 3-25-'25; 3-27-'25.

Pigs from Sow No. 4

No. of pig	Date of autopsy	Results	Remarks
4	4-3-'25	Negative	This pig was stillborn. The lungs, liver, spleen, and intestinal tract were examined for *Ascaris*.
—	—	—	The 9 remaining pigs were not killed and examined for *Ascaris*. At the age of 23 days each of these pigs was treated with oil of chenopodium and castor oil. No worms were expelled by these animals.

TABLE VI (Experiment No. 14)

Sow No. 5—Control

Date of birth	Bred	Date farrowed	No. of pigs in litter	Remarks
3-10-'24	11-17-'24	3-11-'25	5	All pigs were born in a good state of health.

Pigs from Sow No. 5

No. of pig	Date of autopsy	Results	Remarks
1	3-12-'25	Negative	This pig was killed 12 hours after birth. The lungs and intestinal tract were examined for *Ascaris*.
2	"	"	This pig was killed 12 hours after birth. The lungs and intestinal tract were examined for *Ascaris*.
3	3-25-'25	"	The lungs and intestinal tract were examined for *Ascaris*.
4-5	—	—	These animals were not examined.

TABLE VII (Experiment No. 14)

Sow No. 6—Control

Date of birth	Bred	Date farrowed	No. of pigs in litter	Remarks
3-11-'24	11-18-'24	3-13-'25	12	All pigs were born in a good state of health.

Pigs from Sow No. 6

No. of pig	Date of autopsy	Results	Remarks
1	3-13-'26	Negative	This pig was killed at birth. The lungs, liver, and intestinal tract were examined for *Ascaris*.
2	3-14-'25	"	This pig died. The lungs and intestinal tract were examined for *Ascaris*.
3	3-24-'25	"	This pig was killed. The lungs and intestinal tract were examined for *Ascaris*.
4	5- 5-'25	"	This pig was killed. The stomach and intestinal tract were examined for *Ascaris*.
5	5- 5-'25	2 *Ascaris* found	This pig was killed. The stomach and intestinal tract were examined for *Ascaris*. The *Ascaris* found in the intestine measured 40 and 52 mm. in length. Apparently was a result of a spontaneous infection after birth.
—	—	—	The remaining animals were not examined for *Ascaris*.

The two control sows farrowed 17 pigs, 8 of which were examined for *Ascaris*. One of these pigs was found to contain two small *Ascaris* which apparently resulted from a spontaneous infection after birth.

From the data presented above it seems evident that intrauterine infection in swine with *Ascaris lumbricoides* is very uncommon, and if this phenomenon occurs at all, in nature, it must be looked upon as being nothing more than a biologic curiosity.

THE RELATION OF AGE TO ASCARIS INFECTION

It is a well known fact that man and swine may harbor *Ascaris* at any age, but children and young pigs are more commonly infested with this parasite than are adults. This fact may be less manifest in localities where the ascarids are comparatively rare or where 100 per cent of the population are infected. Pantin (69) in a systematic examination of the stools of hospital inpatients in the Dong Kan Hospital, Fuh Kien, China, established the fact that 100 per cent of the population of that particular locality harbored the *Ascaris lumbricoides*. Stewart (97) presented data which show that in some parts of the world *Ascaris* infection is very common in the human being at all ages. Nedergaard (65) examined 3,945 stools of patients in the United Fruit Company Hospital, Preston, Cuba, and found that 33.9 per cent of them were infested with *Ascaris*. In the United States the *Ascaris* is apparently less common in man than in the tropical countries according to the observations made by Moss and Stiles. Moss (64) examined the feces of 547 adult patients from seven southeastern states and found 6.3 per cent to be infested. Stiles (in Moss, 64), in connection with the Amebiasis survey on 8,028 persons representing 23 states, found 89 persons infested with *Ascaris*, or 1.1 per cent. Stewart (97), however, reports that *Ascaris* infestation may occur in as high as 42.8 per cent of the population in some parts of the south. In the Philippines, Garcia (35) examined the stools from 1,603 persons of various ages and

TABLE VIII

Percentage of persons infested with *Ascaris* according to decades

Decade of life	Number of individuals examined	Infested	
		Number	Per cent
First	98	42	42.85
Second	355	119	33.52
Third	615	145	33.54
Fourth	241	65	26.75
Fifth	164	42	25.61
Sixth and Seventh	129	33	25.58

After Garcia (35)

noted that the highest per cent of infestations occurred among persons in the first decade, and the lowest per cent of infestation was observed in those representing the fifth, sixth, and seventh decades.

The reason for the greater frequency of *Ascaris* infestation in children may be the fact that, on account of their habits, they are more commonly exposed to infection than older persons; and also, that they are more susceptible to infection than adults. In swine the amount of exposure to contamination is the same in both young and old animals, altho older animals should have a greater opportunity to become infested than young ones, for the reason that they have lived longer. Thus if animals are equally susceptible at all ages, the older swine should be more commonly infested and have a larger number of *Ascaris* than young ones, but this does not seem to be the case. There appears to be a greater frequency of infestation in young pigs, and also a larger number of parasites are generally found in the young animals. Therefore, it seems evident that younger pigs are more susceptible to infection than older ones. The most complete evidence on this subject was presented by Ransom and Foster (78). The data presented by these writers were obtained by examining the intestines of 2,583 hogs of various ages slaughtered at the Chicago abattoirs. From the evidence it appears that the highest percentage of parasites was found in the intestines of pigs between the ages of two and one-half and five months, and that about half of these pigs were infested. The percentage of pigs infested under two and one-half months of age was less than that of those over that age. Beyond five months of age the percentage of infestation gradually decreased until among hogs over one and one-half years of age only about one-third of the pigs were infested. The average number of worms per pig was also less.

It was also shown that less than three per cent of the worms found in the pigs under two and one-half months of age were adults. In pigs two and one-half to five months of age, nearly 50 per cent of the parasites were mature. In the pigs more than five months of age, over 50 per cent of the worms were adults, and 67.8 per cent of the worms found in hogs from one and one-half to four years of age were found to be mature.

Stewart (99) made observations on 370 pigs which had been killed in one of the English abattoirs, and found that 16.75 per cent of these animals were infested with *Ascaris*.

In conclusion it may be said that man and pig are susceptible to *Ascaris* infestation at any age, but that in human beings, children are more susceptible than adults, and in swine young pigs are considerably more susceptible to infection than older swine.

LONGEVITY OF LARVAE IN THE EXTRACORPOREAL POSITION

It has been observed that *Ascaris* larvae, like those of other nematodes, will survive for a short period outside of the host. Stewart (88) pointed out that larvae will live on moist bread for 24 hours and 48 hours in fresh meat (rat lungs). He (89) noted, further, that larvae that had made the vasculo-pulmonary circuit and reached the large intestine of mice, if placed in tap water were alive and active after 2 hours but were found to be dead at the end of 24 hours. Ransom and Foster (78) kept larvae, from the lungs of a rabbit that had died 10 days after infection with pig *Ascaris* eggs, alive in physiological salt solution for 13 days.

Lungs were taken from a guinea pig that had died 9 days after infection with the eggs of the pig *Ascaris*. These lungs were placed in a refrigerator and after a period of 5 days the writer found them to contain active, living larvae.

That larvae may survive as long as 13 days after their removal from the host, and that they may remain alive in the lungs of a guinea pig for 5 days, would seem to lend some support to Stewart's rat and mouse theory; but the fact that larvae are so slightly resistant to unfavorable conditions, such as dryness, etc., is evidence very much opposed to the possibility of rats and mice acting as intermediate hosts as was suggested by Stewart. Still, the fact that the writer was able to produce many intestinal *Ascaris* in pigs by the feeding of rabbit and calf lungs which contained pig *Ascaris* larvae (Experiment No. 9) is evidence that pigs may become infested with intestinal *Ascaris* by eating mice or rats which have become naturally infected by ingesting *Ascaris* eggs. However, in nature, this method of infestation can by no means be considered a common one.

THE RELATION OF THE HUMAN AND PIG ASCARIS

The probable identity of the large roundworm from the intestine of man and the corresponding intestinal parasite of the pig have in the past few years brought about considerable study by a number of investigators. From the evidence presented, it appears that these parasites are morphologically

and biologically indistinguishable. Bakker (4) studied the two parasites in every detail, morphologically and by means of immunity reactions, and was unable to find any characteristic or constant differences. Schwartz (83) made a comparative study on the biological relationship by means of immune rabbit serum, which was prepared by injecting rabbits with salt solution extract of *Ascaris* from man and swine. Schwartz found that extracts of *Ascaris* from man and swine were not distinguishable so far as the precipitation test was concerned. Ransom and Foster (78) state that the human and pig ascarids are morphologically indistinguishable and probably are identical. Thornton (101) made a morphologic comparison between numerous specimens of ascarids and found that no essential difference exists between the *Ascaris* of man, swine, and chimpanzee, and concludes that his observations conformed with those of Baylis and Daubney (1922) on the *Ascaris* of man, the orang, swine, and Indian squirrels.

A study of the chromosomes in the human *Ascaris* eggs made by Barker (6) showed that the size and shape and the chromosome complex were the same as those described for the *Ascaris* in swine by Edwards (22) and others. The number of chromosomes present was 48, the same as found in the pig *Ascaris* egg.

The question of host relations of these parasites has also received consideration recently by several investigators. Ransom and Foster (78) conducted feeding experiments on several pigs with human *Ascaris* eggs, which resulted in a fatal pneumonia in one pig. The remaining pigs were autopsied at different intervals following the feeding of the eggs, and in each case the intestines were found to be free of *Ascaris*. In 1921 Reiche (80) reported that transmission experiments, where human *Ascaris* eggs were administered to swine, were to be looked upon as positive. This report, however, does not agree with the findings of Ransom and Foster (78), Koino (46), and Paine, Ackert, and Hartman (70). Koino's (46) observations as reported by him show that infectious eggs from the pig *Ascaris* will, when administered to a human being, produce severe pulmonary symptoms; but the larvae will not settle down and develop to maturity upon arrival in the small intestine.

The investigations made by Paine, Ackert, and Hartman (70) were of two kinds, the first phase of the work being the feeding of embryonated human *Ascaris* eggs to pigs, and of pig *Ascaris* eggs to primates, while the second consisted of

studies of the incidence of human and pig ascarids in Trinidad.

The feeding experiments of these authors, in which infectious human *Ascaris* eggs were administered in repeated doses to five pigs ranging from 36 to 48 days of age, caused all of these animals to develop respiratory and other systemic disturbances; while five control pigs remained in a good state of health, and were found to be free of ascarids upon autopsy. The pigs fed embryonated eggs were examined from 54 to 102 days after feeding and were found to be free of ascarids in each case. The three adult primates (two human subjects and a monkey) that were administered pig *Ascaris* eggs by these investigators did not show evidence of infection in that the fecal examinations remained negative from 5 to 22 months after the ingestion of *Ascaris* eggs of porcine origin.

The epidemiological study made by these writers in Trinidad tend to show that the incidence of the human and the pig *Ascaris* are very different. They found that in one of the principal hog raising regions on the island the incidence of human *Ascaris* was 64.3 per cent while only 10.8 per cent of the swine showed evidence of infestation. The results of their experiments led them to conclude that the embryonated eggs of the human *Ascaris* will not produce mature *Ascaris* in pigs, and that infectious pig *Ascaris* eggs will not produce mature *Ascaris* in the adult primates (man and monkey), and that there is thus a physiological difference between the human and pig *Ascaris*.

The limited amount of work reported on the probable identity of the human and pig *Ascaris* by ingestion caused the writer to undertake a few feeding experiments in which infectious human *Ascaris* eggs were fed to pigs.

The data pertaining to these experiments are as follows:

EXPERIMENT NO. 15

In this experiment 14 pigs were used which were 53 days of age at the time of the first feeding of *Ascaris* eggs. Six pigs received human *Ascaris* eggs, 4 received pig *Ascaris* eggs, and the remaining 4 were used as controls for both the human and the pig *Ascaris* egg feedings. These animals were farrowed in concrete stalls which had not been occupied by swine for at least four years.

Examinations of the feces of all these animals were made prior to the feeding of *Ascaris* eggs, by the Vajda (104) method, for *Ascaris* eggs, and in each instance the feces were found to be free of ova, but for safety sake, all animals were given two successive treatments (May 19, 1925, and May 22, 1925) with oil of chenopodium and castor oil. The first medication caused one animal to eliminate two small

TABLE IX (Experiment No. 15)

Pig No.	Fed infectious human *Ascaris* eggs 1925						Fed cavia organs infected with migrating larvae	Fecal examinations 1925						Remarks
	5-25	5-27	5-29	5-30	6-1	6-2	6-2-'25	5-25	6-10	6-17	6-24	7-10	Date of autopsy	
1	X	X	X	X	X	X	—	—	—	—	—	—	8-3-'25	The entire digestive tract was examined and found to be free of *Ascaris*.
2	X	X	X	X	X	X	—	—	—	—	—	—	8-3-'25	The entire digestive tract was examined and found to be free of *Ascaris*.
3	X	X	X	X	X	X	—	—	—	—	—	—	8-3-'25	The entire digestive tract was examined and 1 male *Ascaris* was found to be present in the small intestine which measured 165 mm. x 2.5 mm.
4	X	X	X	X	X	X	—	—	—	—	—	—	8-3-'25	The entire digestive tract was examined and 1 male *Ascaris* was found to be present in the small intestine which measused 173 mm. x 2.5 mm.
5	X	X	X	X	X	X	X	—	—	—	—	—	8-3-'25	The entire digestive tract was examined and found to be free of *Ascaris*.

TABLE IX (Experiment No. 15)—(Continued)

Pig No.	Fed infectious human *Ascaris* eggs 1925						Fed cavia organs infected with migrating larvae 6-2-'25	Fecal examinations 1925					Remarks
	5-25	5-27	5-29	5-30	6-1	6-2		5-25	6-10	6-17	6-24	7-10	Date of autopsy	
6	X	X	X	X	X	X	X	—	—	—	—	—	8-3-'25	The entire digestive tract was examined and found to be free of *Ascaris*.
7	—	—	—	—	—	—	—	—	—	—	—	—	8-3-'25	The entire digestive tract was examined and 1 male *Ascaris* was found to be present in the small intestine which measured 145 mm. x 2.5 mm. Control.
8	—	—	—	—	—	—	—	—	—	—	—	—	8-3-'25	The entire digestive tract was examined and found to be free of *Ascaris*. Control.
9	—	—	—	—	—	—	—	—	—	—	—	—	8-3-'25	The entire digestive tract was examined and found to be free of *Ascaris*. Control.
10	—	—	—	—	—	—	—	—	—	—	—	—	8-3-'25	The entire digestive tract was examined and found to be free of *Ascaris*. Control.

NOTE:—The fact that *Ascaris* eggs were fed is indicated by X. The dash (—) is used to show that the eggs were not administered. The dash (—) used under fecal examinations indicates that no *Ascaris* eggs were found. The X under the heading "Fed cavia organs infected with migrating larvae" is evidence that these animals were fed infected cavia organs.

TABLE X (Experiment No. 15)

Pig No.	Fed infectious pig *Ascaris* eggs 1925						Fecal examinations 1925					Date of autopsy	Remarks
	5-26	5-27	5-29	5-30	6-1	6-2	5-25	6-10	6-17	6-24	7-10		
1	X	X	X	X	X	X	—	—	—	—	—	8-4-'25	The entire digestive tract was examined and found to be free of *Ascaris*.
2	X	X	X	X	X	X	—	—	2 eggs X pres.	—	—	8-4-'25	The entire digestive tract was examined and 1 female *Ascaris* was found to be present in the small intestine which measured 202x3.5 mm.
3	X	X	X	X	X	X	—	—	—	—	—	8-4-'25	In this animal 73 ascarids were found to be present in the small intestine, 48 being females, having a length of from 182-267x 2.5 to 3 mm. in diameter. The remaining 25 male *Ascaris* measured from 127-180 mm. in length by 2.0-2.5 mm. in diameter.
4	X	X	X	X	X	X	—	—	—	—	—	8-4-'25	One female *Ascaris*, measuring 260x4 mm., was found to be present in the small intestine.
5	—	—	—	—	—	—	—	—	—	—	—	8-4-'25	Control. This pig was found to contain 1 male *Ascaris* in the small intestine which measured 145 mm. x 2.5 mm.
6	—	—	—	—	—	—	—	—	—	—	—	8-4-'25	Control. The entire digestive tract was examined and found to be free of *Ascaris*.
7	—	—	—	—	—	—	—	—	—	—	—	8-4-'25	Control. This pig was found to be entirely free of *Ascaris*.
8	—	—	—	—	—	—	—	—	—	—	—	8-4-'25	Control. No *Ascaris* were found to be present in the digestive tract of this animal.

ascarids, while the second dose of oil of chenopodium did not cause any more to be voided with the feces.

The eggs used in this experiment were tested to determine whether or not they were infectious by feeding to two cavias human *Ascaris* eggs and two cavias pig *Ascaris* eggs. The cavias receiving the human *Ascaris* eggs succumbed seven days after receiving the eggs and both animals showed an extreme pneumonia. The lungs of these guinea pigs were heavily infested with *Ascaris* larvae, which indicated that the eggs were infectious. The organs of these animals were fed to two of the pigs which received the human *Ascaris* eggs. The cavias which were fed the pig *Ascaris* eggs died six days after receiving the eggs, and they also showed an extreme pneumonia. The lung tissues likewise contained very many larvae, therefore indicating the infectiousness of these eggs.

TABLE XI (Experiment No. 16)

Pig No.	7-14-'25 Fed infectious human *Ascaris* eggs	7-20-'25 Fed cavia organs infected with migrating larvae	7-14-'25 Fecal examinations	Date of autopsy	Remarks
1	X	—	—	9-16-'25	The entire digestive tract was found to be free of *Ascaris*.
2	X	—	—	9-17-'25	The entire digestive tract was noted to be free of *Ascaris*.
3	X	X	—	9-17-'25	The entire digestive tract was examined and one female *Ascaris* was found in the small intestine which measured 230 mm. in length by 4.5 mm. in diameter. Perhaps present in the intestine or in process of migration at beginning of experiment.
4	X	—	—	9-16-'25	This animal's entire digestive tract was examined and found to be free of *Ascaris*.
5	—	—	—	9-16-'25	Control. — This animal was found to contain two immature ascarids in one small intestine. One, a female, measured 130x2 mm., and the male measured 110x2 mm.

The human *Ascaris* eggs were cultured in a 0.5 per cent formaldehyde solution at approximately 32° C. for 42 days. The pig *Ascaris* eggs were also cultured in the same strength of formaldehyde. These eggs were subjected to a temperature of 32° C. for 36 days, after which the eggs were kept at a temperature ranging from 20° to 26° C. for 77 days.

The pigs given eggs in this experiment received them in very small, repeated doses so as to prevent an extensive tissue reaction by large masses of migrating larvae at the same time, and also to more nearly approach infestation as it supposedly occurs in nature.

Further details of this experiment may be found in Tables Nos. 9 and 10.

Table 9 contains data pertaining to the feeding of human *Ascaris* to swine.

The data incorporated in Table 10 pertain to the feeding of pig *Ascaris* eggs to swine.

EXPERIMENT NO. 16

In this experiment five pigs were used, four of which received human *Ascaris* eggs in a large, single dose; the fifth animal was used as a control.

The animals used in this experiment were also treated with oil of chenopodium and castor oil.

The eggs used in this experiment are the same as the human eggs used in Experiment No. 15 except that they were incubated 49 days longer at the temperature of from 20° to 26° C.

At the time this experiment was started the eggs were again tested to determine their infectivity on one cavia. This cavia developed a severe pneumonia, and *Ascaris* larvae were demonstrated in the bronchial mucus. The organs of this animal were fed to one of the experimental pigs.

Further data pertaining to this experiment may be found in Table 11.

Ascaris infestation in man and swine in hog raising districts of Nebraska apparently seem to have no relation to each other in that there is a certain amount of *Ascaris* infestation in practically all herds of swine, while human *Ascaris* infection appears to be very uncommon.

The evidence presented by other authors, and the results obtained by the writer in the above mentioned experiments, indicate that the *Ascaris* of man and swine are morphologically and biologically indistinguishable, but physiologically separate species; that is, the *Ascaris* larvae from human embryonated eggs will not develop to maturity in swine and the infectious pig *Ascaris* eggs will not produce mature ascarids in the human being. This condition is comparable to the findings of Mégnin (in Ewing, 24) in regard to several of the species of the Sarcoptidae.

The *Ascaris lumbricoides* may occasionally be found to settle down in the intestine of aberrant hosts and then apparently

develop to a point approaching the adult stage. The *Ascaris* observed in sheep is thought to be nothing more than an imperfectly developed *Ascaris lumbricoides*. Rudolphi (82) reported, under the name of *Ascaris ovis*, one female specimen in the Vienna Museum collection. This specimen was later described by Diesing (20) and von Drasche (21), and the latter also described two badly preserved specimens of *Ascaris* found in sheep by Koebel. Copeman (11) in 1841 noted the small intestine of a lamb to harbor 75 small ascarids which he identified as *Ascaris lumbricoides*. Neumann (67) observed 29 male and female ascarids in a sheep and identified them as *Ascaris ovis*. The sexual organs were found to be similar to those of *Ascaris lumbricoides*. He was unable to find ripe eggs in the females.

Ransom and Foster (78) report that the Bureau of Animal Industry collection contains several specimens of *Ascaris* collected from sheep at Brookings, South Dakota, Blairville, Pennsylvania, and Bethesda, Maryland. They (77) also report the finding of 50 partly grown *Ascaris* in an experimental lamb that was artificially infected with pig *Ascaris* 103 days previous to findings. They also twice artificially infected a young goat with the eggs of pig *Ascaris*. The animal was examined 27 days after the first feeding, and thousands of *Ascaris*, about 10 millimeters in length, were found to be present in the small intestine. The writer administered eggs of the pig *Ascaris* to two young goats. Symptoms of pneumonia followed, but an examination of the intestine of one of the goats, nine months later, showed it to be free of ascarids.

The fact that there has been no case where fully developed female *Ascaris* were found in sheep, together with the experimental evidence presented by Ransom and Foster (77), should justify the common belief that the so-called *Ascaris ovis* is merely the pig *Ascaris* in an abnormal host.

Recently Schwartz (84) reported the finding of a large number of *Ascaris lumbricoides* in a 5-months-old calf which was slaughtered at the National Stock Yards, Illinois. He noted that a number of those parasites had reached a length of 110 mm. and also that fertile eggs were present in some of the females.

It has also been observed that other ascarids will live in a strange host. Evidence of this kind has been reported by Jammes and Martin (44), who found that in the intestine of man, immature *Ascaris vitulorum* developed from eggs which were taken from the female of the species.

The evidence presented warrants the conclusion that the human and pig *Ascaris* are distinct species, but that this parasite may occasionally be found to develop to partial maturity in the intestines of animals other than their definite hosts.

It is here proposed to specify the type of *Ascaris lumbricoides* by the variety names *hominis* and *suis*.

PARASITIC ALLERGY

In recent years it was discovered that parasitic extracts caused local reactions seemingly of an allergic nature when brought in contact with the mucous membrane of the eye of various animals.

It was found by Weinberg and Julien (106) that the introduction of perienteric liquid of the *Ascaris megalocephala* into the eyes of 220 horses caused about two-thirds of them to show a positive reaction. The reactions varied in their intensity. In a number of animals, only an edema and congestion of the conjunctive were noted, while some of the other horses presented pronounced systemic disturbances. The horses used by these investigators were autopsied and examined for evidence of parasites. The examinations showed that 52 of the animals harbored *Ascaris* at the time of the autopsy. They also found that only 16 of these animals showed positive reactions. Van Es and Schalk (105) also observed that the introduction of an extract of *Ascaris megalocephala* into the eye produced in many instances a marked reaction and in some cases a pronounced systemic disturbance.

Ransom, Harrison, and Couch (79) reported that a marked general reaction was noted following the introduction of fluid from the body cavity of *Ascaris lumbricoides* into the conjunctival sac of hogs.

Perienteric fluid and an extract of pig *Ascaris* were used by the writer in experiments similar to those mentioned above, but the results were somewhat different.

EXPERIMENTS ON PARASITIC ALLERGY

The following experiments were designed to determine, if possible, whether an allergic sensitization exists in pigs that are or have been infested with *Ascaris*.

EXPERIMENT NO. 17

In this experiment 19 shoats were used. Six had been treated subcutaneously with *Ascaris* extract.[1] Six were infested with *Ascaris*.

[1] The extract was prepared by crushing adult *Ascaris* in a mortar and macerating for 24 hours in ten times their weight of 50 per cent glycerin. The mixture was then centrifuged and filtered.

The remaining seven pigs were raised as worm-free pigs and were not treated subcutaneously with an *Ascaris* extract.

The eye test was made May 24, 1921, by instilling into one eye of each pig a few drops of the *Ascaris* extract by means of a camel's-hair brush. Observations were made from time to time and no evidence of a reaction was noted in any of the pigs up to 24 hours.

May 25, 1921, the pigs were again tested in the same eye with a few drops of the extract. There were no reactions produced in any of the pigs.

EXPERIMENT NO. 18

In this experiment the extract was introduced into the right conjunctival sac of 26 young pigs (September 29, 1924), some of which were known to be infested with *Ascaris*.

Of this number, two pigs reacted. One of the pigs showed a slight reaction, that is, there was a moderate redness of the conjunctiva and a mucous discharge. The second showed a decided reaction. This reaction manifested itself by an extreme redness of conjunctiva and a marked swelling of both eyelids. There was also a pronounced mucous discharge. The reaction in both cases subsided in less than three hours.

EXPERIMENT NO. 19

December 31, 1925. Twenty-five adult hogs were inoculated with an *Ascaris* extract into the left conjunctival sac. There was not the slightest evidence of a reaction in any of the animals.

EXPERIMENT NO. 20

In this experiment 5 pigs (shoats) which were infested with *Ascaris* were used.

June 2, 1921. An *Ascaris* extract[1] was instilled into the left subjunctival sac of each animal. No reactions were noted in any of the pigs.

EXPERIMENT NO. 21

On September 25, 1921, perienteric fluid originating from the body cavity of the pig *Ascaris* was inoculated into the left conjunctival sac of five pigs which were infested with *Ascaris*. Not the slightest reaction followed the instillation.

EXPERIMENT NO. 22

In this experiment 25 pigs (shoats) were similarly inoculated with a perienteric fluid (September 29, 1924). Six pigs showed evidence of an allergic reaction; of these animals three showed only a slight reaction. The remaining three pigs had marked reactions. The reactions in each case occurred within a few minutes and in course of three hours all traces of the reaction had disappeared.

EXPERIMENT NO. 23

January 12, 1926. Twenty-one adult hogs were likewise inoculated with a perienteric fluid, which originated from the body cavity of the pig *Ascaris*. Of these animals six showed an allergic reaction. Five of the swine showed a mild reaction, that is, there was a moderate edema of the conjunctiva covering the eyelid and nictitans membrane. The sixth animal showed a slight redness and edema of the mucous membrane of the eyelid.

[1] The extract used in experiment No. 20 differs from that used in No. 17 in that it was prepared with physiological salt solution instead of the 50 per cent glycerin.

EXPERIMENT NO. 24

January 13, 1926. In this experiment 52 medium-sized shoats were each inoculated with a similar fluid. Of these animals 30 showed evidence of an allergic reaction. Four of the 30 animals showed a very slight reaction (a very slight redness and edema of the conjunctiva covering the eyelid and nictitans membrane). Nineteen reacted slightly (slight redness and edema of the conjunctiva covering the eyelid and nictitans membrane, and also a slight watery discharge). The remaining 7 pigs showed a moderate reaction (pronounced edema and redness of the mucous membrane of the eyelid and nictitans; and also a marked watery discharge). The reactions in these pigs all appeared within a few minutes after inoculation and disappeared in less than four hours. Several days after the pigs had been treated with the perienteric fluid, 3 of the animals were killed and their intestinal tracts were examined for *Ascaris*. Two of these animals which did not show any evidence of a reaction harbored mature *Ascaris*. The small intestine of the one contained 36 mature worms, while the second contained only two. The third pig showed a moderate reaction and the small intestine contained 25 medium-sized *Ascaris*.

The pigs used in the above experiments were, with the exception of the pigs used in Experiment No. 17, all exposed to *Ascaris* infection thruout the greater part of their lives; and some of the animals included in each group were known to have been infested with *Ascaris*.

It is evident from the results obtained in the above experiments that *Ascaris* infected pigs do not react allergically to the same extent that worm infested horses do. It is also apparent that a negative finding is no indication that the pig was not infested with *Ascaris* at some time during its life.

IMMUNITY

It is a well known fact that old animals are not, as a rule, heavily infested with parasites and are usually more resistant to parasitic invasion than young animals. This fact, together with other observations, led a number of investigators to suggest that resistance in animals against *Ascaris* infection was probably due to previous infections with this parasite. Stewart (88) suggested that there was a possibility of one infection preventing a subsequent one. He also stated that he had never observed worms of obviously different ages associated together in the intestines of pigs. Yoshido (108) conducted a few experiments on animals which had been previously infected with *Ascaris* larvae. The results obtained in these experiments did not permit definite conclusions. Ransom and Foster (78) indicate that they are not certain as to what causes the lessened susceptibility of pigs against *Ascaris;* but they suspect that age is the determining factor

rather than an acquired immunity as a result of a previous infection. They state that, if an immunity is established as a result of a previous infection, it is not acquired immediately, and cite as evidence an experiment on a kid in which they showed that two infections may occur 17 days apart. On the other hand, Fülleborn (28) seems to think that previous infections may produce a certain degree of immunity against a subsequent infection. Hadwin (39) advances the theory that immunity against ascarids is stimulated and increased by repeated attacks of these parasites. He also suggests that in addition to the production of anti-substances to neutralize the cast-off products of worms, there is another substance secreted by the eosinophiles which is detrimental to the worms themselves.

To determine, if possible, whether the greater resistance of old animals toward *Ascaris* infection is due to previous parasitic invasions, the writer undertook the following experiments:

EXPERIMENT NO. 25

In this experiment three male pigs (about 10 weeks old) originating from a lot of pigs which were rather heavily infested with *Ascaris* were each given five doses of an *Ascaris* extract (the extract was prepared the same as that described in Experiment No. 17) as set forth below:

February 28, 1921. Each pig received 5 c.c. each of the *Ascaris* extract subcutaneously. Immediately after the inoculation there was a moderate anaphylactic reaction. All of the animals recovered within 20 minutes after the first symptoms were noted.

March 4, 1921. Each pig received subcutaneously 4 c.c. of *Ascaris* extract (a small desensitizing dose preceded the 4 c.c. dose). One of the pigs showed a slight anaphylactic reaction.

March 12, 1921. The three male pigs each received 3 c.c. of *Ascaris* extract subcutaneously. A very slight transitory reaction occurred in one animal.

March 17, 1921. Two c.c. of *Ascaris* extract was administered subcutaneously to the three pigs. A slight reaction was noted in one of the pigs.

March 22, 1921. Each pig received one c.c. of *Ascaris* extract subcutaneously. Slight reactions were noted in two pigs.

April 22, 1921. The above pigs together with three female untreated ones, which were used as controls, were each given a suspension of infectious pig *Ascaris* eggs.

April 24, 1921. One of the treated pigs was found to be dead. The cause of this pig's death was an acute pneumonia. The various organs were examined, and were free of *Ascaris* larvae.

April 29, 1921. Extensive *Ascaris* pneumonia caused the death of a control pig. Many *Ascaris* larvae were noted in the respiratory organs, and adult *Ascaris* were found in the small intestine.

The remaining four pigs presented marked symptoms of pneumonia, which was, in all probability, caused by the migration of *Ascaris* larvae.

May 5, 1921. The remaining pigs showed improvement.

May 9, 1921. The remaining pigs, with the exception of a treated one, had completely recovered.

June 3, 1921. The only pig which had not recovered by May 9 was found to be dead. On autopsy it was noted that pneumonia, due to the migration of *Ascaris* larvae, was the cause of death. No ascarids were found in this animal.

July 8, 1921. The three remaining pigs were killed and autopsied. One adult *Ascaris* was found in the small intestine of the male pig. In one female pig, four adult *Ascaris* were noted in the small intestine. The small intestine of the remaining female pig contained 11 *Ascaris*, of which two were adults. The adults measured 32 cm. in length and 0.5 cm. in diameter. The smallest immature ascarid measured 5.5 cm. in length and 0.1 cm. in diameter.

EXPERIMENT NO. 26

In this experiment 13 pigs were used. They originated from two litters and were born March 16, 1921 (3 males and 3 females), and March 18, 1921 (4 males and 3 females). All of the female pigs were injected with an *Ascaris* extract, and the male pigs were used as controls.

The inoculations of the extract were made as follows:

March 21, 1921. Each of the six female pigs received subcutaneously 5 c.c. of the *Ascaris* extract. Five of these animals received a desensitizing dose of 0.05 c.c. of *Ascaris* extract ten minutes before the subcutaneous injection. All of the female pigs showed a reaction (excitement, squealing, vomiting, pruritis, and some incoordination of movements). The reaction in some cases lasted no longer than five minutes, and at the end of a half-hour all of them appeared normal.

March 24, 1921. Each one of the six females received a subcutaneous injection of 4 c.c. of *Ascaris* extract after receiving a desensitizing dose of 0.05 c.c. Aside from a tendency to scratch themselves, the injections caused no reactions.

March 28, 1921. Each of the six females received a subcutaneous inoculation of 3 c.c. of *Ascaris* extract 10 minutes after the administration of a desensitizing dose of 0.05 c.c. Only one of the pigs showed a slight reaction (squealing and pruritis).

March 31, 1921. Each female pig received a subcutaneous injection of 2 c.c. of *Ascaris* extract, after a desensitizing dose of 0.1 c.c. was administered. No reactions were observed.

April 4, 1921. Each one of the six female pigs received subcutaneously 1 c.c. of the *Ascaris* extract, after receiving a desensitizing dose of 0.1 c.c. No reactions were observed.

May 10, 1921. All of the pigs (males and females) were immunized against hog cholera by the simultaneous method.

June 9, 1921. All of the pigs (13) received a large dose of infectious *Ascaris* eggs per orum.

June 14, 1921. All of the pigs showed respiratory symptoms except one female and two males. These three pigs presented symptoms two days later.

June 16, 1921. Two of the female pigs were found to be dead. The cause of death in both cases was a very extensive *Ascaris* pneumonia.

The respiratory organs contained very many larvae. Petechiae in the kidneys of one pig were also found to contain larvae. There were three immature *Ascaris* noted in the small intestine of each animal. These parasites measured from 4.3 cm. to 17.0 cm. in length.

The 11 remaining pigs showed very pronounced respiratory symptoms.

June 17, 1921. One female was found to be dead, as the result of a very extensive *Ascaris* pneumonia. The respiratory organs contained very many larvae.

June 18, 1921. Another female was found to be dead as the result of a very extreme *Ascaris* pneumonia. Many larvae were present in the respiratory organs. Twenty-two *Ascaris* measuring from 4.4 cm. to 10 cm. in length were noted in the small intestine.

June 19, 1921. One male died. This animal's death was caused by an extensive *Ascaris* pneumonia. Many larvae were found to be present in the respiratory organs and the digestive tract.

June 25, 1921. The respiratory symptoms disappeared in the eight remaining pigs, but all these animals were in a decrepit condition. Four of the male pigs were discarded without an autopsy at this time.

September 22, 1921. The remaining females and the two males were again given a large dose of *Ascaris* eggs. Four days later one of the females was found to be dead, due to a chronic pneumonia probably originating from the previous invasion of *Ascaris* larvae. The small intestine contained 51 immature *Ascaris*.

September 29, 1921. One of the male pigs showed very marked respiratory symptoms. This animal was killed six days later, at which time 15 adult *Ascaris* were found in the small intestine. The two remaining pigs did not show evidence of lung invasion.

October 31, 1921. Both of these animals were destroyed. On autopsy 12 adult *Ascaris* were found in the small intestine of the female, but none were noted in the male.

EXPERIMENT NO. 27

The object of this experiment was to ascertain whether one or more injections of *Ascaris* extract have any influence on the development and further distribution of *Ascaris* larvae which may be introduced several days after the inoculation of the extract.

In this experiment, 39 cavias were injected with the extract according to the following table:

TABLE XII

No. of cavias inoculated	Injected 1921		
	3-12	3-15	3-18
5	1.0 c.c.	0.5 c.c.	0.25 c.c.
6	—	0.5 c.c.	0.25 c.c.
6	—	—	0.25 c.c.
6	—	—	0.5 c.c.
6	—	—	1.0 c.c.
10	Controls		

May 23, 1921. All of the above cavias were fed a small quantity of *Ascaris* eggs, which proved to be non-infectious.

June 9, 1921. Each of the 39 cavias was fed a suspension of infectious *Ascaris* eggs. On the following day one of the cavias was found to be dead from some unknown cause.

On June 13, 1921, one of the cavias was found to be dead and the remaining animals all showed marked symptoms of pneumonia. Ten days after the ingestion of eggs all of the cavias but two were dead as a result of *Ascaris* pneumonia. The respiratory organs from a large number of the animals were examined and in each instance they were found to contain very many larvae. One of the surviving cavias was not injected with the *Ascaris* extract, and the other received a single 1 c.c. dose of the extract.

July 29, 1921. The two cavias which recovered from one *Ascaris* infection were again fed *Ascaris* eggs. Seven days after the feeding of eggs both animals were sick.

August 5, 1921. One of the cavias was found to be dead and the other one died the following day. Both animals showed a very pronounced pneumonia on autopsy. The lungs and trachea were swarming with larvae.

From the results obtained in the foregoing experiments on immunity, it is apparent that subcutaneous inoculations with *Ascaris* extract do not immunize pigs and cavias against *Ascaris* infections. It is also evident that one or more infections with *Ascaris* do not immunize against subsequent infections.

It is apparent that the reason why swine become resistant to *Ascaris* infection is not definitely known; but from the results obtained by the writer it appears that this resistance is due to an age factor and not to an immunity, at least not an immunity comparable to the one in microbic infections.

GENERAL SUMMARY

1. It has been observed that the uteri from pig *Ascaris* as small as 125 mm. in length may contain eggs which will develop to the infectious stage when incubated at the proper temperature.

2. Adult male *Ascaris* may attain a length of 310 mm. and females a length of 490 mm.

3. Eggs subjected to a temperature of 31° C. to 34.5° C. in the presence of moisture and oxygen may develop to the vermiform stage in ten days. Segmentation of the eggs does not take place if they are subjected to a temperature of — 5° C. to 10° C.

4. Eggs placed at a temperature of from — 5° C. to 10° C. in a dry condition were found to be viable after two years. Eggs kept at this temperature in a moist condition remained viable for over four years, at which time they were still

capable of developing to the vermiform stage and producing *Ascaris* pneumonia in the cavia.

5. Hatching of *Ascaris* eggs occur chiefly in the small intestine. The hatching of eggs may occur in the alimentary tract of a guinea pig as early as two hours after ingestion. Hatching of the eggs does not seem to take place when introduced beneath the skin of pigs.

6. Investigations show that the larvae migrate thru the circulation from the intestine to the lungs by way of the liver and heart, thus confirming data presented by Stewart and Ransom and his associates.

7. Evidence has been obtained which shows conclusively that *Ascaris* larvae, after having completed their vasculopulmonary circuit in pigs, will settle down and develop to maturity in the small intestine of this animal.

8. Pigs may become infested with intestinal *Ascaris* as a result of ingesting tissue which is infected with *Ascaris* larvae. This evidence seems to indicate that rodents can act as intermediate hosts. However, it is quite obvious that this method of infection is by no means a common one in nature.

9. In pigs, *Ascaris* may develop to maturity as early as 49 days after the ingestion of infectious eggs.

10. Contact with rabbits which had been fed *Ascaris* eggs produced *Ascaris* pneumonia in normal rabbits, thus indicating a possibility that rodents may act as intermediate hosts.

11. Pathologic changes may be observed in almost any organ in the body as a result of migrating *Ascaris* larvae.

12. Intrauterine infection in swine with *Ascaris* appears to be very uncommon and if this phenomenon occurs at all in nature, it must be looked upon as being nothing more than a biologic curiosity.

13. It is apparent that the *Ascaris* of man and swine are morphologically and biologically indistinguishable, but physiologically are distinct species; that is, adult *Ascaris* will not occur in the small intestines of swine from the ingestion of human *Ascaris* eggs and vice versa.

14. *Ascaris* infected pigs do not react allergically to the same extent that worm infested horses do. It is also certain that a negative reaction is by no means an indication that the pig was not infested with *Ascaris* at some time during its life.

15. It appears that the resistance acquired by pigs against *Ascaris* infection is due to an age factor and not to an immunity, at least not an immunity comparable to the one in microbic infections.

ACKNOWLEDGMENT

The writer is deeply indebted to Dr. H. Busman, inspector in charge of meat inspection station at Omaha, Nebraska, and his associates, and to Dr. W. C. Foltz, Lincoln, Nebraska, for the collection and shipment of pig *Ascaris* material; also to Professor N. Nitta, Imperial Institute for Infectious Animal Diseases, Nishigahara, Japan, for the supply of human *Ascaris* material, which was used in this work.

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